HUMAN GENETICS

FRITZ LENZ

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Human genetics

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Outline of Human Genetics and Racial Hygiene

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Volume I:

Human Genetics

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Prof. Dr Erwin Baur, Prof. Dr Eugen Fischer and Dr Fritz Lenz, Fritz Lenz

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Introduction.

The composition of every nation is constantly changing. The people who inhabited Rome at the end of the Imperial period were completely different, not only in their customs and habits, but also in their
overall character and racial characteristics, from the people of Cato's time. And the people living in Germany today are, in their mental and physical racial characteristics, entirely different from the contemporaries of Charlemagne. This further development, this transformation of a people, can lead to its betterment, to its strengthening and to its advancement; but it can also—and this is the most common case among all civilised peoples—bring about, more rapidly or more slowly, its decline and degeneration.

It has often been believed, in a thoroughly unfounded comparison of a people with an individual human being, that every civilised people goes through, as it were, a fresh stage of youth, a stage of maturity and finally a stage of old age, i.
i.e. degeneration and decline. That is surely not the case. But in every nation, pathological processes—mostly processes of natural selection—can set in, which rapidly deteriorate the character of the nation and bring about its decline.

The realisation that such processes are manifesting themselves to an alarming degree in our nation, as in all civilised nations, and that we, like all civilised nations, are in the throes of degeneration, is already widespread today. Attempts are also already being made to counter this 'disease of the nation as a whole' with socio-political and legislative measures, though mostly, of course, with rather ineffective means.

Just as scientific medicine is impossible without a thorough knowledge of human anatomy, physiology and pathology, so too is the study of human sociology,

any purposeful population policy and for all eugenic endeavours (eugenics), a broad foundation in the natural sciences is essential.

If we do not know which different racial components make up a people, according to which laws the races differ and the countless differences between individuals are inherited , and how selection processes affect a population, we are in the dark about all considerations regarding the effect of social and political influences on the creativity of a people in complete darkness. Legislative interventions in the field of population policy and racial hygiene undertaken without this knowledge would be to be regarded in the same light as the quackery of an uneducated layman, which is a danger to the public.

This consideration forms the basis for the structure of our textbook. The first section provides an overview of the general theory of variation and heredity. The second section deals with the specific theory of human variation and heredity, including anthropological racial differences. The third section then discusses the most important hereditary diseases and anomalies, and the fourth deals with the hereditary basis of intellectual ability. Taken together, these four sections form a coherent whole, in that they provide a theoretical account of all the factors on which an individual's predisposition depends. For readers who are less interested in the practical implications these have for our social and personal lives, we have compiled these theoretical sections into a separate volume, available for purchase on its own. The main practical focus, however, lies in the second volume. There, the processes of natural selection taking place within our populations are first described, with the conclusion that the hereditary constitution of our population is currently changing, on the whole, in an unfavourable direction. The final sections then deal with practical racial hygiene; that is to say, they examine how this degeneration might be halted and how the life of the race might be steered in the most favourable direction possible. In particular, they also

how the individual and the individual family can organise their lives in accordance with the principles of practical racial hygiene.

Section One.

An Outline of the General Theory of Variation and Heredity

By Professor

Erwin Baur.

1. Some Basic Concepts.

By ‘inheritance’ in the biological sense of the word, we generally mean the fact that the offspring of a parent, or—in the case of sexual reproduction—of a pair of parents, resemble that parent or those parents

.

Offspring can arise in multicellular organisms:

1. through the separation and independent further development of a single cell, or in organisms with multinucleated cells, even a part of a cell;
2. through the separation and independent development of entire groups of cells and organs;
3. through the separation of two cells from a single individual or from two individuals, followed by the union of two such cells (‘egg cell’ and ‘spermatozoon’) to form a single cell, from which a new individual then develops.

The first two cases are referred to as a s e x u a l l e s s (vegetative) form of reproduction, whilst the third case is referred to as a s e x u a l (sexual) form of .

In a great many organisms, particularly among plants, any cell or group of cells can be separated from its original structure. It then begins to grow independently , thereby giving rise to a new individual[1]. In many other organisms, only a few cells are ‘capable of regeneration in all directions’ in this way. In yet other organisms, particularly in higher animals, generally only very specific cells — the gametes — are capable of producing new individuals.[2]

In the organisms of the first two groups, therefore, each of these fully regenerative body cells must contain everything essential to the species in question; in the organisms of the last group, at least each germ cell must contain everything essential to the species in question. The subtle structure of the egg cells of an orangutan and a human must, in some way, account for the entire difference between an orangutan and a human. Of course, one must not imagine this in the

In the simplistic terms of the preformation theory, one might imagine that all the differences that will appear in the later organism are already present in the egg cell. Every fully-formed organism is merely the end result of a long individual development; minute differences at the initial stage can lead to profound differences in later stages — small causes, great effects. — It is quite easy to imagine that small differences in the structure of two egg cells that are outwardly indistinguishable—for example, minor chemical variations—might result in one developing into a European and the other into a Hottentot. Nor is it at all certain that there is a definite relationship between the magnitude of the differences between two germ cells and the magnitude of the differences between the fully formed organisms.

We shall now, without initially attaching any cytological notions to it, refer to that part of a cell in which its species-specific character is established—in a manner as yet unknown to us—using a term introduced by Naegeli as **'idioplasm'**. According to this definition, all hereditary differences between two fully developed individuals are ultimately attributable, in terms of developmental mechanisms, to differences in the structure or chemistry of the idioplasm. These idioplasm differences are the primary ones; the differences visible externally in the fully grown organisms are something quite secondary. Some minor difference between the idioplasms of two egg cells may well be the developmental cause of a whole series of differences in the most diverse parts of the fully formed organism.

The inheritance from a parent or a pair of parents to their offspring is based on the fact that the offspring have, wholly or in part, the same idoplasm as the parent or, in the case of as the parents.

2. The manifestations of variation.

Inheritance is almost never perfect; that is to say, the offspring are almost never completely identical to the parent or parents, and likewise, siblings almost always differ from one another. It is said that the offspring 'v a r i e'. It is the most important task of genetics to clarify the laws, according to which this difference, this variation, is anticipated.

We recognise essentially three causes for the differences between children and their parents, and between children themselves—that is, causes of variation—and thus three groups of variations can also be distinguished:

1. One of these three main causes lies in the fact that, even in terms of hereditary predisposition, i.e. i.e. idioplasmatically, completely identical individuals may differ greatly depending on the external conditions under which they have grown up, depending on their 'peristase'. We refer to this type of variation as variation through modification or, using a new term adopted by convention in this book, as **paravariation**.

2. A second cause of variation lies in the fact that, in sexual reproduction, a new individual almost always arises through the union of two cells which differ in their genetic endowment, i.e. h. differ in their idoplasm, and that such a mixing results in a combination of two directions of inheritance. This is then referred to as **mixovariation**.

3. A third cause of variation lies in the fact that, for whatever reason and at whatever point in time, a change occurs in the structure of the in the generally very stable idioplasm, and that this results in the emergence of cells—and consequently individuals—with altered idioplasm, which are correspondingly altered in their secondary external characteristics. Variations based on such a change in the idioplasm are often referred to as mutations; we shall refer to them as **idiovariations**.

a) The Paravariation.

In order to investigate the laws governing paravariations, one must begin with cases where the various causes of variation do not come into play simultaneously, and where, above all, the effects of the sexual mixing of two hereditary tendencies are ruled out. We therefore choose, for the purpose of ly deriving the most important laws, an organism that reproduces continuously or at least temporarily without the sexual union of different idioplasms. We must therefore work either with organisms that reproduce asexually or with organisms that reproduce 'autogamously'[3]. The latter is the case, for example, with beans, which have hermaphroditic flowers, and in which the pollen almost always reaches only the stigma of the same flower and fertilises it. Individuals that are idioplasmically identical, which have arisen through a s e x u a l reproduction of a parent individual, are referred to in the literature on inheritance as a **'clone**'. Individuals that are genetically uniform in the same way, which are obtained as the offspring of a parent individual in the case of an autogamous organism, are referred to as **a 'pure line**'.

Let us take as a textbook example a culture of *Paramecium caudatum*, a small single-celled infusor commonly found in all puddles and pools, which can be propagated purely vegetatively in small glass vessels over a long series of generations. Thus, if one introduces a single parent organism into an aquarium, one quickly obtains a large swarm of organisms which are all identical in terms of their hereditary disposition, i.e. h. i d i o p l a s m a t i c a l l y i d e n t i c a l. Externally, however, the individual organisms of such a clone are nevertheless very different, because each one has developed under slightly different conditions. One, for example, has always received sufficient food, whilst another has gone hungry at times; one has sustained an injury, whilst another has not; one was situated in a particularly well-lit or particularly warm part of the culture vessel, and so on.

Very specific laws apply to this diversity among genetically identical organisms, which is caused by the unequal external influences acting upon the animals: if one statistically examines any given characteristic of all the animals in such a colony, one usually finds that individuals deviating significantly in one direction are rare, as are those deviating significantly in the other direction. However, the closer the expression of the characteristic approaches the mean, the more frequent the individuals in question are. This is illustrated very clearly by Table I.

which presents statistical data on body length for a clone of *Paramæcium*. Figure 1 shows the same data in the form of a variation curve.

Table I:

Length in μ	136	140	144	148	152	156	160	164	168	172	176	180	184	188	192	196	200
Number of animals of this length	2	5	5	26	14	27	40	52	39	32	26	14	12	3	2	1	

It is easy to see why the variation curves so often take precisely this form, i.e. more or less resemble the binomial curve: the size of the paramecia, to stick with this example, is influenced by all sorts of different factors. For example, an animal will only grow particularly large if it has been consistently well-nourished, has never been injured, has always been in water with sufficient oxygen, has never been harmed by excessively bright light, or by temperatures that are too high or too low , and so on. Only an animal that is fortunate in all these respects will grow particularly large, and likewise, only an animal that is unfortunate in all these respects will be particularly small. Most often, however, it will be the case that an animal is partly fortunate and partly unfortunate, d. h. it will usually be of an intermediate size.

Nevertheless, this whole question is so important that it is worth deriving why this particular shape of the curve arises.

Size distribution of the animals

Fig. 1.

There are countless factors that influence the size of a paramecium in an aquarium. Let us focus solely on those mentioned earlier and assume that for each of these five factors there are always only two alternatives: one that promotes size and one that inhibits it. Let us also denote the promoting alternatives with a capital letter and the inhibiting ones with the corresponding lower-case letter. This would give the following:

Promoting alternatives	Inhibiting alternatives
A Good nutrition	*a* Poor nutrition
B sufficient oxygen	*b* Insufficient oxygen
C Suitable lighting	*c* Light too bright

D no damage caused by cold	*d* temporary damage caused by cold
E no damage caused by heat	*e* temporary damage caused by heat

Even if we take into account only these five independent factors, the individual animals in a population can develop under as many as **32** different conditions. An animal that grows up under the exclusively favourable conditions A B C D E will become particularly large; an animal that grows up under the only partially favourable conditions A b c D e – that is, one which is well-nourished and does not suffer from the cold, but which is damaged by a lack of oxygen, heat and excessively bright light – will be considerably smaller. For the sake of simplicity, let us assume that each favourable factor causes an animal to grow by one unit of length; we then have the following:

Possible combinations of the five independent factors	Degree of growth experienced by an animal raised under this combination
ABCDE	5
ABCDe	4
ABCdE	4
ABCde	3
ABcDE	4
ABcDe	3
ABcdE	3
ABcde	2
AbCDE	4
AbCDe	3
AbCdE	3
AbCde	2
AbcDE	3
AbcDe	2
AbcdE	2
Abcde	1
aBCDE	4
aBCDe	3
aBCdE	3
aBCde	2
aBcDE	3
aBcDe	2
aBcdE	2
aBcde	1
abCDE	3
abCDe	2
abCdE	2
abCde	1
abcDE	2
abcDe	1

abcdE	1
abcde	0

All 32 possible combinations have the same probability, so one can expect that, out of a large number of animals in an aquarium, 1/32 will be found in one of these . However, as a glance at the table on p. 10 shows:

1 combination	a	increase	of	5
5 Combinations	"	"	"	4
10 "	"	"	"	3
10 "	"	"	"	2
5 "	"	"	"	1
5 Combination	"	"	"	0.

In other words, we must expect that, out of a large number of animals

1/32	a	increase	of	+5
5/32	"	"	"	+4
10/32	"	"	"	+3
10/32	"	"	"	+2
5/32	"	"	"	+1
1/32	"	"	"	+0

will be shown. In the form of a curve , this results in the diagram shown in Fig. 2.

Distribution curve

Fig. 2.

If, in this example, we had chosen a larger number of factors – say, 6 – instead of 5, we would have obtained the sequence **1 6 15 20 15 6 1**; and any larger number of factors would likewise yield sequences such as

1
1 2 1
1 3 3 1
1 4 6 4 1
1 5 10 10 5 1

etc. correspond to the coefficient values of $(a+b)^n$, i.e. they correspond to the Gaussian probability curve.

The characteristic shape of the paravariation curve therefore stems solely from the fact that a great many factors influence the paravariation quite independently of one another.

Fig. 3.
Two littermates from an otherwise very uniform breed of pig.
The animal on the left is barely fed, whilst the one on the right is well-fed
(according to S. v. Nathusius).

The variation curves obtained when one statistically examines any characteristic very often show a more or less faithful reflection of the random curve, but by no means always. There are also single-peaked, multi-peaked
and other such curves. This is because the change in a characteristic does not necessarily have to run exactly p a r a l l e l to the changing conditions. However, it would take us too far afield to go into these questions in more detail.

How strikingly large differences arise between individuals that are genetically identical, and how immense the extent of paravariation can be, has indeed been established in numerous experiments on plants; however, for animals—and above all for higher animals—little reliable data is available on this subject. It is very difficult to obtain genetically uniform material in this context, because, due to sexual reproduction, offspring can only be obtained from two individuals. A very instructive experiment of this kind involving higher animals is shown in Figure 3.

Fig. 4.
Schematic representation of a selection experiment within a *Paramaecium* clone. Both the largest and the smallest organisms in the initial culture produce the same offspring. Organisms of the same size are shown stacked on top of one another within each culture to illustrate the frequency of the individual size classes.

When an organism, through the influence of the environment or through its 'peristase', underg oes a paravariation, this results in a change to its idoplasm, its hereditary disposition is generally unaffected. To illustrate this, we would do best to return to our Paramecium example:

If we rear a clone of Paramaecium in an aquarium, the individual organisms vary greatly in size, but both the smallest and the largest organisms are genetically 'plasmatically' entirely identical; they both produce the same offspring, which in turn consist of both large and small individuals (Figure 4).

Even if such selection on the basis of 'large or small' is carried out over many generations, the result remains the same, t h e s p e c i f i c c h a r a c t e r i s t i c s of **the individual animal** are **not** inherited; **rather, each animal in this lineage always passes on only the very specific, characteristic modifiability of its lineage**.

It is also possible — in a slightly different experimental setup — to transfer one animal from a clone into a nutrient-rich aquarium and another into a nutrient-poor one. This will result in schools developing in the two aquariums that are v e r y different. The well-nourished school will, on a n a v e r a g e, consist of larger animals than the poorly nourished one. The two schools will be very different, even though they are genetically identical. One can continue these breeding lines in the two different aquariums for a long series of generations, and if one then selects one animal from the 'well-fed' line and one from the 'underfed' line, and places these two animals in aquariums of exactly the same conditions, both will produce shoals that are exactly alike. The idioplasm of the clone has therefore not been altered by these very different culture conditions, however different the two strains may have been over many generations.

In the case of *Paramecium caudatum*, a whole host of different strains can easily be fished out of any pond; each of these has its own specific modifiability, which it inherits quite faithfully, and represents a very specific **idiotype**.

All this applies quite generally, as can perhaps best be illustrated by a botanical example: Of the common Chinese primrose — the well-known ornamental plant — there are many colour varieties, including a red and a white one, which we shall designate as Primula sinensis rubra and Primula sinensis alba respectively. Like all characteristics, flower colour is subject to very strong paravariation due to all manner of external influences, such as temperature. If plants from a uniform 'red' lineage are grown in a warm, somewhat shaded greenhouse at around 35°C, they flower white, whilst their siblings grown at around 10–15°C are red. The offspring of such plants—which have developed white flowers in the warm greenhouse and are indistinguishable from ***Primula sinesis alba***—will have red flowers if grown under normal conditions, i.e. at 10–15°C. The hereditary, idioplasmic difference between the two races is therefore not that one has 'white' flowers and the other 'red' ones, but that the two strains react differently to temperature.

To the layman, Paraffinum durum and Paraffinum liquidum differ in that one is a solid white mass at normal temperature, whilst the other is an oily liquid. However, if both paraffins are heated to a temperature of 60°C, they are just as indistinguishable in appearance as the two primrose varieties in the greenhouse. Nevertheless, the two heated and outwardly indistinguishable paraffins are, of course, just as

different as the two primrose varieties in the greenhouse. The difference between the two paraffins is precisely that they have different melting points, i.e. they react differently to changes in temperature. Just as little as a 'paraffinum' when heated to 60°, and by having a lower melting point, h. it becomes 'Paraffinum liquidum', just as a **Primula sinensis rubra** that blooms white in a greenhouse does not become a **Primula sinensis alba**.

To emphasise this once more, a characteristic distinguishing a species is not a specific manifestation of a trait, but always and only a specific 'mode of reaction'. What an organism looks like therefore always depends on two things: firstly, on its inherited mode of reaction—or, one might also say, on its idioplastic constitution—and secondly, on its peristase, that is, on the external influences to which it was subjected during its development.

It is quite remarkable how many vague notions on this subject are in circulation.

The notion that the change—the paravariation of an external characteristic, such as the colour of the primrose— also automatically alters the hereditary response pattern of the lineage, persists in many people's minds on the basis of misinterpreted observations under the catchphrase of 'the linkage of acquired characteristics' still haunts many minds.

The only remedy here is a completely clear definition of the term. A heritable trait is always and only a 'heritable, specific mode of reaction' to external influences. The emergence of a new trait therefore depends on this earlier mode of reaction being altered. If one does not merely heat paraffinum durum to 60°, i.e. does not merely melt it, but instead subjects it to very high temperatures under pressure, then its chemical constitution changes; under certain circumstances, a paraffin with a lower melting point may then be produced from it. Similarly, through some extraordinary influence—such as radium irradiation, prolonged cultivation at a high temperature just within the plant's tolerance, and the like, a Prunula sinensis rubra may produce offspring that react to temperature in a different way to the parent strain with regard to their flower colour.

However, just as the liquefaction of Paraffinum durum brought about by a change in its chemical constitution and a lowering of its melting point is something quite different from mere melting, so too is the

formation of such a new primrose variety is, in principle, quite different from the simple paravariation achieved through cultivation in a greenhouse.

As we heard earlier, a change of the latter kind is referred to as an idiovariation or, to use an older technical term, a mutation.

The term 'acquisition of a new characteristic' is, unfortunately, still used by many biologists to refer to these two entirely different phenomena.

So if we wish to comment on the delicate question of the inheritance of acquired characteristics, we will have to say:

Newly arising characteristics, including those produced as a result of any external influences, which are indeed **new** characteristics in the sense just mentioned, are heritable, or at least may be heritable, whereas the fact that an organism undergoes a paravariation does not automatically imply that an idiovariation also arises. The fact that the notion is so firmly entrenched in many people's minds—that a paravariation always, or at least usually, triggers an idiovariation of more or less the same nature—stems from incorrectly conducted experiments and from erroneous conclusions.

Fig. 5.
8 different clones of *Paramaecium*. The line X—X indicates the mean size of all 8 clones. The symbol '+' denotes the mean size for each individual clone (according to Jennings).

The greatest source of error lies in genetically heterogeneous experimental material. The error is so common, and is made so often, that it is worth discussing it here using an example must be. As mentioned earlier, there are a number of different strains of *Paramaecium caudatum*, each of which is genetically stable. A number of such *Paramaecium* strains are shown in Fig. 5. For each of these eight strains, the pattern shown in Fig. 4 applies: each individual does not pass on its own size, but rather its strain-specific character. As a glance at Fig. 5 shows, the paravariation curves of the different strains overlap. So if one has these different Paramecium clans mixed together in the same aquarium, one finds a completely continuous range of animals from very large to very small; one does not notice that there are eight genetically distinct strains (clones) in the aquarium. Even if one knew this, it would be impossible to tell from any single animal of medium size to which clone it belongs. Animals of this size are found in all eight clones. The fact that two animals are 'paratypically' identical does not mean that they

are also 'idiotypically' identical . If one were to carry out a selection experiment with such a mixture, as shown in Figure 4, selecting the smallest animal on the one hand and the largest on the other, one would nevertheless find that the largest animal has, on average, a larger offspring than the smallest animal. As a glance at Figure 5 shows, the largest animal is certainly one from the lineage depicted at the top; whilst it does not pass on its individual size, it does, however, pass on its lineage-specific character, i.e. there will be offspring that are, on average, larger than the original mix. Similarly, the smallest animal will be one from the lineage shown at the bottom and will pass on its average small size. So if, in this case, the selection of animals deviating significantly from the average results in a shift in the average size in the direction of the selection applied, this is no proof that the paravariation is in any way heritable, but merely the consequence of the fact that the initial material is non-uniform. Here too, the animals that deviate significantly do not pass on an acquired paravariation, but only their inherited ancestral trait. This source of error plays a very detrimental role in the literature on the inheritance of acquired characteristics.

A second source of error in this discussion is that, very often, external influences which have acted upon an individual also influence its offspring to a greater or lesser extent, albeit indirectly.

Such after-effects can very often be observed. If a large number of plants are grown from a pure line of beans, the individual plants vary in size and vigour depending on nutritional conditions, etc. However, here too, only the characteristic of the lineage is inherited; that is to say, selecting large or small plants within such a pure line does not result in any change in the average size.

What applies to the paramecia shown in Figure 4 also applies to this bean experiment. One can, however, allow a bean plant to half-starve and wither away, so that it develops just a few shrivelled and small seeds. The seeds will then produce seedlings which, in the early stages of their development— when they are surviving solely on the reserves passed on by the mother —are very poorly nourished. Consequently, they develop into significantly weaker bean plants than the offspring of a plant of the same line that has not been mistreated and has been grown under otherwise identical conditions. It is therefore clear that there is no 'inheritance of the individual paravariation' of the mother plant here; it is merely the same unfavourable nutritional conditions, which had already affected the mother, also continued to affect her offspring

their earliest stage of development. One generation later, the after-effects in the beans are balanced out.

Far more striking are similar after-effects of paravariations in higher animals. This is understandable when one considers that, in all viviparous animals, the embryos initially undergo the m o s t s u b s t a n t i a l p a r t o f t h e i r d e v e l o p m e n t w h i l e i n t h e w o m b. Furthermore, however, in many animals the eggs themselves are already, so to speak, 'pre-developed' to a great extent; many developmental processes have already been initiated, and certain 'organ-forming substances' and 'organ-forming regions' have formed within the egg plasma. Thus, a large part of the development takes place in the womb and is therefore also influenced by external conditions affecting the mother.

It is precisely this source of error that played a major role in the much-cited experiments by Kammerer, not a single one of which is even remotely free from flaw.

A l l experiments on the heritability of paravariations, in which these and all other sources of error have been avoided, have unequivocally shown that such heritability d o e s n o t o c c u r.

It is, of course, possible that chance might one day cause one and the same cause to trigger both a specific paravariation and an idiovariation of the same type; so it would, for example, be possible that the cultivation of *Primula sinensis rubra* in a very warm greenhouse might both induce a paravariation of the flower and bring about a change in the idioplasm. It is also possible that, by chance, the result of this idiovariation might be a variety which produces white flowers even at low temperatures. However, not a single case of such a coincidence is known, and it is quite clear that the probability of this ever occurring is very small. In any case, there is absolutely no question of this connection between paravariation and idiovariation occurring frequently, let alone being the rule!

The above-mentioned laws apply to all organisms examined in this regard. We must expect that human beings are no exception. However, there is no observational data on this subject for human beings that is even remotely reliable.

All observations of everyday life show, however, that the physical and mental characteristics of a fully developed human being are also the result of two factors: firstly, their inherited predisposition, i.e. their inherited idioplasmic constitution, and secondly, the external influences—such as diet, upbringing, etc.—under which they have developed. It is highly probable that climatic influences, in the broadest sense of the word, also trigger paravariation in humans. The fact that the

people of a nation—which, whilst representing a very diverse racial mixture, nevertheless live together in a specific region—frequently exhibit certain shared physical and mental characteristics that are difficult to describe, is probably, to a t e il, connected with this.

In all studies of heredity in humans, too, we must always bear in mind that an individual does not pass on the specific development of their personal characteristics, but rather their own inherited predisposition. What is inherited, for example, is not an inguinal hernia itself, but 'a certain predisposition to develop an inguinal hernia'. Whether a person with this predisposition actually develops an inguinal hernia also depends on other factors. However, for the purposes of further inheritance, it is entirely irrelevant whether such a person with a genetic predisposition develops a hernia — for example, as a result of severe exertion or the like — or not. Even a person who has remained outwardly healthy passes on the predisposition. In the case of a hereditary predisposition to a disease, where the onset of the clinical picture is triggered only by a not overly frequent combination of external factors, it is, as just mentioned, very difficult to trace the hereditary lineage precisely. If, for example, a moderate predisposition to hernias is inherited within a family, only a few of the individuals affected by this hereditary condition who subject themselves to any particular strain or the like will actually develop a hernia. When examining the family tree of such a family, one very often gets the impression of a completely irregular, 'capricious' pattern of inheritance, even though in reality it is a very regular pattern of inheritance — but not of personal disposition, rather of 'predisposition'.

It must also be borne in mind that every human being undergoes a very large part of their development in the womb, and that environmental influences affecting the mother can therefore still have far-reaching consequences for the next generation. One must therefore always expect 'after-effects' (see page 19).

A child born to a woman with no family history of tuberculosis, but who has contracted severe tuberculosis—perhaps as a result of her occupation—and who, because of her tuberculosis, is in the worst possible state of nutrition, will very often be frail and less resilient from birth and will therefore be particularly susceptible to contracting tuberculosis themselves. Thus, a hereditary predisposition may be mistakenly assumed where it is in fact merely a secondary effect. In a case that appears outwardly quite similar, where a woman with a hereditary predisposition to tuberculosis — due to a particular shape of the thorax, a certain deficiency of the lymph glands or some other factor — develops tuberculosis and then also

gives birth to children with tuberculosis, there is not only an after-effect but also a hereditary link. In such a case, distinguishing between the two is very difficult and very often completely impossible. In any event, the utmost caution in all conclusions and the most rigorous critical examination of the material are indispensable in all such investigations.

Just as paravariations are not hereditary in any other organisms, neither are they hereditary in humans. Yet even here, the most bizarre notions are widely held. Whilst it is true that a child without a musical aptitude can, through careful upbringing, be led to a certain understanding of music and to practising it to some extent, the view, widespread in popular literature, that the offspring of such musically trained people are born from the outset with a b e t t e r musical aptitude than their parents—and that it is therefore possible simply to enhance hereditary aptitude through education—is completely unfounded. Although upbringing has a strong influence on the individual, it does not affect the hereditary predisposition of offspring. A people or a social class that is inferior according to its hereditary predisposition — such as the Black people in the United States of North America — is, through education and the influence of culture, raised to a higher level in its individual — but this does not alter the race as such.

b) The mix-variation.

In sexual reproduction, the genetic makeup of the two parents will usually differ to a greater or lesser extent. The case in which the two gametes coming together are genetically completely identical is likely to occur almost exclusively in self-fertilising organisms, i.e. those hermaphroditic plants and animals in which the gametes of both sexes from the same individual always unite. However, if the two gametes uniting are not genetically identical, this results in a very complex pattern of inheritance and is the source of those phenomena of variation which we term m i x o v a r i a t i o n s.

Fig. 6.
Crossing of an ivory-coloured (a) with a red (b) variety of the garden snapdragon (*Antirrhinum majus*). The hybrid (c) is pale red; the offspring of the hybrid, produced by self-pollination or by cross-pollination between two such hybrids, consist of pure red (d), pale red (e) and ivory-coloured plants in the ratio of 1 red : 2 pale red : 1 ivory.

To understand the principles at work here, let us once again start with a simple case. Let us, for example, cross two plants of *Antirrhium majus* (the garden snapdragon): an ivory-coloured one (Fig. 6a) from a pure ivory-coloured strain and a red one (Fig. 6b) from a pure red strain. We shall introduce specific letter designations for these two plants: each plant arises from the union of two gametes. Let us denote a gamete from a pure red strain by **F,** and the red plant resulting from the union of two such gametes by **FF**. Similarly, the gametes of the ivory-coloured plant shall be called **f** and the plant itself **ff**. If we produce a hybrid between a red plant **FF** and an ivory-coloured **plant ff** by allowing a female gamete **F** to fuse with a male **gamete f**, or—which amounts to the same thing—a female gamete **f** to fuse with a male **gamete F**, we obtain a plant designated **Ff**, or **fF**, i.e. a hybrid, or, to use the technical term, a heterozygous, ‘non-uniform’

(arising from the union of dissimilar gametes) individual. In contrast, an organism that has arisen from the union of two similar gametes is called homozygous (of the same genotype). Such an Antirrhinum hybrid will now produce flowers that are neither ivory-coloured like one parent, nor red like the other, but rather pale red (Fig. 6c). It has inherited the 'ability to produce red flower colour' from only one — the red — parent, and this manifests itself in the fact that it exhibits a considerably paler colour. So far, there is nothing unexpected about this; what is all the more striking, however, is the behaviour of the offspring of such a hybrid: if we allow a number of hybrids to cross-pollinate amongst themselves, or if we—which can be done without harm—pollinate one of them with its own pollen, then we obtain offspring consisting of three different types of plant. Some of these hybrid offspring have red flowers, just like one of the parent varieties; a second group has ivory-coloured flowers like the other parent variety; and a third group has pale red flowers like the original hybrid. If one cultivates a large number of such descendants of the original crossed plants, it is easy to observe that the three groups—the red, pale red and ivory-coloured plants—occur in a ratio of 1 : 2 : 1; that is to say, out of 100 plants, approximately 25 will be red, 50 pale red and 25 ivory-coloured. The actual figures obtained from an experiment involving 97 plants, for example, are 22 red, 52 pale red and 23 ivory-coloured. The red plants obtained in this way produce exclusively red offspring, whilst the ivory-coloured ones produce only ivory-coloured offspring, but the p a l e r e d individuals behave e x a c t l y l i k e t h e o r i g i n a l h y b r i d **Ff**, i.e. they split again into ¼ red, ²/₄ pale red and ¼ ivory-coloured offspring. The experiment can be represented in the form of a pedigree as follows:

Pedigree, page 25

The letters printed on the right-hand side here, as in Fig. 6, are used to denote P_1, F_1, F_2, $F_{(3)}$ denotethe following: P_1 is the first parental generation, i.e. the individuals originally used for the cross; F_1 is the first filial generation, i.e. the

Primary hybrids, F_2, F_3 , etc., are the subsequent generations of hybrids. Correspondingly, $P_2$is the parent generation of P_1, and so on.

An understanding of all these peculiar phenomena of segregation, this 'alternative inheritance', is provided by the theory established by Mendel, which is now generally accepted, that each such hybrid produces two types of gametes, namely 50 per cent 'paternal' and 50 per cent 'maternal'. According to this theory, our Antirrhinum hybrid **Ff** produces two types of egg cells and pollen grains; half of these are precisely of the type **F**, behaving exactly like the gametes of a homozygous FF plant, whilst the other half are exactly the same as the gametes **f** of the ivory-coloured race **ff**. If we pollinate the hybrid with its own pollen, or if we allow several such hybrids to cross-pollinate one another, then the two different types — **F** and **f** — of gametes can combine in four different ways:

> An egg cell **F** may be fertilised by a pollen grain **F**, producing a plant **FF** that flowers red; an egg cell **F** may be fertilised by a pollen grain **f**, producing a plant **Ff** that flowers pale red; an egg cell **f** may be fertilised by a pollen grain **F**, producing a plant **fF** that flowers pale red; an egg cell **f** may fuse with a pollen grain **f** to produce a plant **ff** that flowers in an ivory colour.

All four possible combinations have the s a m e probability; we may therefore expect that, in the offspring of such a hybrid, all four will occur with equal frequency, and that, accordingly, these four types of plant—**FF**, **Ff**, **fF**, **ff**—will occur in approximately equal proportions. Theoretically, therefore, it is to be expected that the offspring of such a hybrid will consist of:

¼ plants resulting from **F×F**, i.e. homozygous for red
" " " " **F×f** " " heterozygous pale red
" " " " **f×F** " " " " "
" " " " **f×f** " " homozygous ivory

It has been possible, on the basis of the theory, to predict the results of further experiments and thus to test the theory. This applies in particular to the back-crossing of a hybrid: for example, we pollinate our hybrid **Ff** with pollen from the ivory-coloured parent plant **ff**

or another ivory-coloured plant of the same genus. According to the hypothesis, the hybrid produces two types of egg cells: one half of the egg cells transmits only the trait of red flower colour, whilst the other half transmits only the trait of ivory flower colour. If we were to fertilise such a hybrid with an ff plant, whose pollen grains all transmit only the trait of ivory-coloured flowers, then 50 per cent of the resulting offspring must be produced by the union of an F egg cell with an f pollen grain, and 50 per cent by the union of an f egg cell with an f pollen grain. Consequently, such a backcross must produce: 50 per cent plants of the formula **Ff**, i.e. pale red heterozygotes, and 50 per cent plants of the formula **ff**, i.e. ivory-coloured plants that continue to pass on their pure traits. The result predicted by the theory is indeed borne out; in corresponding experiments, one obtains, in almost equal proportions, on the one hand ivory-coloured plants that remain constant, and on the other hand pale red plants that continue to show Mendelian inheritance.

In the examples given, the hybrids are easily recognisable by their pale red colour. The hybrids thus occupy, as it were, a middle position between the parents; they are 'intermediate' hybrids. Whilst this is very common, it is by no means always the case! The appearance of the heterozygotes can also be quite different.

A particularly common scenario is where the heterozygotes resemble one parent entirely and are 'goneocline'; for example, if we cross a black mouse with a white one, we obtain hybrids that look black and are externally indistinguishable from the homozygous black animals. The $F_{(2)\ \text{generation}}$ of such a cross therefore consists, when viewed externally, of three-quarters black and one-quarter white animals. However, an examination of the offspring of the black animals shows that here too, one part of them is homozygous black, whilst two other parts— —are heterozygous black. Here too, therefore, the proportions are in reality the same as in the case of the hybrids discussed earlier between the red and ivory-coloured snapdragons.

Fig. 7.
Cross between a red peloric (a) and an ivory-coloured normal variety (b) of *Antirrhinum majus*. The hybrid (c) is pale red and normal. The F_1 generation consists of red and

pale-red normal (d), red and pale-red peloric (e), ivory-coloured normal (f) and ivory-coloured peloric (g) plants in the ratio of:

9 + – dark red normal	3 ivory-coloured normal
3 + – " " peloric	1 " peloric

Pure red and pale red are not distinguished in this figure.

This phenomenon, whereby many hybrids are outwardly indistinguishable from one of their parents, is referred to by the term 'dominance'. It is said that one trait – in this case, the 'ability to produce black hair colour' – dominates over the other trait, namely the absence of this ability, and that the latter trait is recessive to the former. This phenomenon of dominance—which is by no means a general rule—has often been attributed unduly great significance, with people speaking of a 'rule of dominance'. This is entirely incorrect; there is no universally applicable rule of dominance, and very often, upon very close inspection, even in cases of apparent complete dominance, it is still possible to distinguish homozygotes from heterozygotes.

It also happens that the dominance of a trait is expressed to varying degrees depending on the age of the hybrids.

Exactly the same principles apply, mutatis mutandis, even when we cross breeds that differ in more than one characteristic – that is, when we produce not merely 'monohybrids' but 'di-, tri- and polyhybrids'. Let us again consider a very simple case here. We cross a red peloric snapdragon variety (Fig. 7a) with a normal-flowered ivory-coloured one (Fig. 7b); the hybrid is pale red and has a completely normal flower shape, though the upper lip is slightly smaller than in the homozygously normal plants. We therefore have a case here of almost complete dominance of the normal flower form over the peloric form, whilst in terms of colour the hybrid occupies roughly a middle position. The F_2 generation (Fig. 7d–g), obtained by self-pollination of such a hybrid, consists of six externally distinct groups of plants, namely:

red normal	(3)	pale red peloric	(2)
pale red normal	(6)	ivory-coloured normal	(3)
red peloric	(1)	" peloric	(1)

The numerical ratios in which these various categories occur are given in ( ). How these categories

and these ratios arise, we shall derive in a similar manner to that used in the example discussed first: we denote a sex cell—or, as is usually stated in the literature on heredity, a g a m e t e —of the normal ivory-coloured race by **fE**, and a sex cell of the red peloric race by **Fe**. Here, the following applies:

F	Ability to produce red flower colour	**f**	Absence of this ability, i.e. the so-called gamete transmits the ivory flower colour
E	Ability to produce normal flowers	**e**	Absence of this ability, i.e. this gamete conveys the peloric flower form.

The single P_1plant resulting from the union of two gametes **F e** (red flower colour, peloric flowers) therefore has the genotype **FF ee**.

The other P_1plant, resulting from the union of two gametes **f E** (ivory-coloured normal flower), then has the genotype **ff EE**.

The hybrid consequently has the genotype **Ff Ee**. When this hybrid reaches sexual maturity, according to Mendel's theory, it produces four different types of gametes, namely

F E, which red colour and normal form
F e, " " " " peloric " "
F e, " ivory " " normal " "
F e, " " " " peloric " "

These four different types of gametes are produced in equal numbers.

If we cross a hybrid such as **Ff Ee** with itself, or if we cross several such hybrids with one another, then the four types of egg cells can combine with the four types of pollen grains in **16** different combinations, namely:

1. an egg cell **FE** may be fertilised by a pollen grain **FE**, producing a normal red plant **FFEE**.
2. " " **FE** " " " " **Fe** and gives a red normal plant **FFEe**.
3. " " **FE** " " " " **fE** and produces a pale red normal plant **FfEE**.

4.	"	"	**FE**	"	"	"	"	**fe** and yields a pale red normal plant **FfEe**.
5.	"	"	**Fe**	"	"	"	"	**FE** and produces a red normal plant **FFEe**.
6.	"	"	**Fe**	"	"	"	"	**Fe** and produces a red peloric plant called **FFee**.
7.	"	"	**Fe**	"	"	"	"	**fE** and produces a pale red normal plant **FfEe**.
8.	"	"	**Fe**	"	"	"	"	**fe** and produces a pale red peloric plant called **Ffee**.
9.	"	"	**fE**	"	"	"	"	**FE** and produces a pale red normal plant **FfEE**.
10.	"	"	**fE**	"	"	"	"	**Fe**, resulting in a pale red normal plant **FfEe**.
11.	"	"	**fE**	"	"	"	"	**fE** and produces a normal, ivory-coloured plant **ffEE**.
12.	"	"	**fE**	"	"	"	"	**fe** and produces a normal, ivory-coloured plant **ffEe**.
13.	"	"	**fe**	"	"	"	"	**FE** and produces a pale red normal plant **FfEe**.
14.	"	"	**fe**	"	"	"	"	**Fe** and produces a pale red peloric plant called **Ffee**.
15.	"	"	**fe**	"	"	"	"	**fE** and produces a normal, ivory-coloured plant **ffEe**.
16.	"	"	**fe**	"	"	"	"	**fe** and produces an ivory-coloured peloric plant **ffee**.

A glance at the overview shows that, of the 16 possible combinations, three (1, 2, 5) plants have red normal flowers, six (3, 4, 7, 9, 10, 13) plants have pale red flowers, one (6) plant with red peloric flowers, two (8, 14) plants with pale red peloric flowers, three (11, 12, 15) plants with ivory-coloured normal flowers, and one (16) plant with ivory-coloured peloric flowers.

We shall therefore obtain in $F_{(2)}$ of this cross the 6 ly different plants:
'red normal', 'pale red normal', 'red peloric', 'pale red peloric',
'ivory normal' and 'ivory peloric' in the ratio 3 : 6 : 1 : 2 : 3 : 1. If we combine red and pale red,
[A] the ratio is 9 : 3 : 3 : 1. The figures obtained in experiments are thus in good agreement; such a cross, for example, produced the following F_2 generation:

Red normal **39**, pale red normal **94**, red peloric **15**, pale red peloric **28**,
ivory normal **45**, ivory peloric **13**.

Furthermore, based on the theory, it is to be expected that one third of each of the normally flowering categories will have offspring that are exclusively normal-flowering, namely all plants containing **EE** (Nos. 1, 3, 9, 11); furthermore, we must expect that two

of these, namely all those containing **Ee** (Nos. 2, 4, 5, 7, 10, 12, 13, 15), will in turn split into normal and peloric offspring; likewise, it is to be expected that all red FF plants will have only red offspring, whereas all pale-red Ff plants must show a split in the next generation into red, pale-red and ivory-coloured plants. This theoretical requirement is also fulfilled in a l l the experiments. This experiment is illustrated in the form of a pedigree diagram opposite.

Pedigree, page 32

We can therefore see that the various characteristics by which the two originally crossed varieties differ are distributed quite independently of one another to the gametes of the hybrid.

A cross corresponding in every respect to the one just discussed, in which only both traits show complete external dominance, is shown in Fig. 8. The cross between a smooth-haired black guinea pig breed and another rough-haired white breed yields, in F_1 produce rough-haired black offspring, whilst in F_2 four different types of offspring appear: black rough-haired, white rough-haired, black smooth-haired and white smooth-haired, in a ratio of 9 : 3 : 3 : 1.

Fig. 8.
Crossing of a black, smooth-haired breed of guinea pig with a white, shaggy one. The F_1 offspring are black and shaggy, and in the F_2 generation, black shaggy, black smooth, white shaggy and white smooth offspring appear in a ratio of 9 : 3 : 3 : 1.

It is also possible to cross breeds that differ in even more characteristics. For example, we can cross an ivory-coloured, normal, tall snapdragon with a red, peloric, short-growing one. The $F_{(1)\ generation}$ is then red and normal, and the generation is tall. Tall growth dominates short growth to a considerable extent. If we denote a gamete that transmits the 'tall growth' trait as **X** and one that transmits the 'short growth' trait as **x**, and retain the previous designations for form and colour, then the 'formula' for the ivory-coloured, normal, tall variety is **ff EE XX** and that for the red, peloric, short variety is **FF ee xx**. The hybrid is then **EeFf Xx** and produces eight different gametes, namely: **FEX**, **FEx**, **FeX**, **Fex**, **fEX**, **fEx**, **feX**, **fex**, between which **64** different combinations are possible. Here too, there are no external differences

all 64 combinations; however, if we disregard the differences between homozygotes and heterozygotes, we find eight different types of plant in F_2, namely: **1.** red normal tall (27), **2.** red normal short (9), **3.** red peloric short (9), **4.** red peloric tall (9), **5.**
ivory normal tall (3), **6.** ivory normal short (3), **7.** ivory peloric tall (3), **8.** ivory peloric short (1). The numerical ratio in which these different plants occur in $F_2$is given in brackets (). Each of these types can be bred homozygously; it is therefore quite possible, through a single such cross-breeding, to obtain a whole series of new varieties, which, strictly speaking, are all merely new combinations of the independently inherited differences between the parent varieties. If we cross breeds that differ in even more characteristics, the same laws continue to apply. The table opposite (p. 35) shows the numerical ratios that arise in this context and the general formula that can be derived for n characteristics.

As the number of independently inherited differences between two crossed breeds increases, the composition of the F_2generation consequently becomes extremely complex very rapidly. If, for example, two breeds exhibit seven independent differences, then as many as $2^7 = 128$ externally distinct types of individuals already appear in F_2.

Table on page 35

Numerous results of crosses, which at first did not appear to conform to the laws of segregation at all, have nevertheless been recognised, upon closer examination, as being entirely consistent with them. Admittedly, a great many complexities in the patterns of segregation have come to light, which can often make understanding a cross very difficult. The most important of these is that an apparently uniform trait always depends on several independently Mendelian ‘factors’. For example, the brown colour in a bird’s feathers may arise because a colourless chromogen only acquires its brown colour through the action of an enzyme. However, the ‘ability to produce the enzyme’ and the ‘ability to produce the chromogen’ may be independently inherited traits, each of which follows Mendelian inheritance independently. For example, a white breed of chicken may lack the ability to produce the enzyme, whilst the ability to produce the chromogen

is present. Another white breed may lack the ability to produce chromogen whilst possessing the ability to produce the enzyme. If two such white breeds, which are white for different reasons, are crossed, hybrids will result which have inherited the ability to produce enzymes from one parent and the ability to produce chromogen from the other, and which consequently, due to
'hybrid atavism' and which, when mated amongst themselves, will continue to produce offspring that are nine parts coloured and seven parts white.

If we carry out numerous cross-breeding experiments with any plant or animal, we soon come to identify a certain number of such hereditary factors, and in doing so it becomes apparent time and again that the seemingly vast number of varieties - and breeds within a single 'species' are **always** based on **different combinations** of a relatively small number of Mendelian differences. This is probably easiest to illustrate using the example of rabbits. The most important and most common coloured breeds can be traced back to the following differences:

Basic difference	Effect.
Aa	All aa individuals are completely unable to develop any coat or eye colour, regardless of their other genetic makeup; they are white with red, i.e. colourless, eyes – 'typical albinos'. **Aa** and AA rabbits, however, which lack the second important pigment factor **X** (and therefore have the genetic formula **XX** or **Xx**), are white with blue eyes (e.g. the 'white Viennese rabbits').
X x	**X** is the second important factor in pigment formation; together with **A** it enables the formation of a yellow coat colour.
B b	**B** enables the yellow coat colour produced by **A** and **X** to be converted into a light brown.
C c	**C** changes the brown colour produced by **A**, **X** and **B** into blue.
D d	**D** makes the hair colour produced by the other factors darker. **AXBcd** is light brown. **AXBcD** is chocolate brown (the which is 'havana-coloured' have this colour). **AXBCd** is blue (like the 'blue Viennese'). **AXBCD** is deep black.

G g **G** means that the hairs are not uniformly coloured along their entire length, but display band-like light and dark zones. An animal with the formula **AXBCDg** is black; one with the formula **AXBCD** is wild-coloured, like wild rabbits. Similarly, an animal with the genotype **AXBCdg** is uniformly blue, whereas one with the genotype **AXBCdG** is 'blue-wild-coloured', i.e. blue in base colour but with the banded coat pattern of wild rabbits. Likewise, there are corresponding 'yellow-wild-coloured' animals, and so on.

Apart from the coat pattern, **G** is also characterised by the fact that all G animals have a white, or at least very pale, belly and a white underside to their tail, whilst all gg animals have a belly and tail underside coloured in the same way as the rest of their body.

K k All K animals are piebald, i.e. they display irregular white patches in their coat, which is coloured according to the rest of their genotype.

M m All mm animals, which according to their other genotype should be black, are black-spotted on a yellow background. Corresponding mm animals, which according to their other formula should be wild-coloured, are wild-coloured-spotted on a yellow-wild-coloured background; and those which, according to their other formula, should be blue, are blue-spotted on a light yellow background, and so on.

N n nn rabbits have the colour determined by the rest of their genotype, but only on the nose, in the ears, on the feet and on the tail. Rabbits with this colouring are called 'Russian-coloured'; the rest of their coat is white and they have red eyes. The colour of their 'peripheral' body parts depends on the rest of their genotype. There are wild-coloured, black, blue, etc. Russian rabbits.

O o **O** also determines a specific distribution of colour across the body. An animal with the formula **AXBCDgkMNo** is p u r e b l a c k; one with the formula **AXCDgkMNO** is 'b l a c k a n d t a n' in colour, i.e.

i.e. black with a tan tinge above the eyes, in the ears, on the paws and in certain other areas of the body (this is a colouring very commonly found in dachshunds, Dobermannpinschers, etc.). O-animals which, according to the rest of their formula, should be blue, are blue and tan, etc.

P p All P-type animals have white guard hairs interspersed throughout their coat, which is coloured according to their other genotype; they are 'silvery'. This silvering, which apparently corresponds to the greying of horses, first appears in animals aged approximately

6 weeks of age. There are several 'factors acting in the same direction' (cf. p. 40), all of which intensify the silvering.

V v All **vv** animals have angora hair.

These twelve basic differences alone, in their various combinations, give rise to a very large number (4096) of breeds, and by crossing animals whose genetic formula is known, one can produce any new combinations—that is, new breeds—as shall be demonstrated by an example.

An animal with the formula **a a X X B B C C D D g g k k M M N N o o p p v v** is **white Angora with red eyes**. An animal with the formula **A A X X B B C C d d G G k k M M NN oo pp VV** is **blue wild-coloured and short-haired**.

The hybrids between these two rabbits are wild-coloured and smooth-coated (and thus resemble wild rabbits exactly). In the F_2 generation, however, a division occurs into the following types: 1. wild-coloured, short-haired; 2. wild-coloured, Angora-haired; 3. black, short-haired; 4. black, angora-haired; 5. blue wild-coloured, short-haired; 6. blue wild-coloured, angora-haired; 7. blue-grey, short-haired; 8. blue-grey, angora-haired; 9. white, short-haired; 10. white, angora-haired. Or to give another example, the cross **between a a X X B B C C d d g g k k M M N N o o p p v v** (white Angora rabbit) **and A A x x B B C C d d G G k k M M N N o o p p V V** (white Viennese rabbit) produces wild-coloured, short-haired animals which, in turn, in F_2 a very varied breakdown. A corresponding case in humans would be the cross between an albino African (fair-skinned) and a light-skinned European, where F_1 is dark-coloured, like other mulattos.

The fundamental differences listed are not the only ones that have been identified in rabbits to date. A whole series of others are already known today, but even so, the number of fundamental differences is incomparablely small when compared with the very large number of racial differences. The **12** basic differences listed alone allow, as mentioned, for **4,096** genetically distinct types, of which, however, a quarter – that is, **1,024** – are white with red eyes and a further 768 are white with blue eyes. All the other **2,304** types, however, also differ in appearance. Of these, only a relatively small number have been bred and established as recognised breeds; yet it is very easy to produce any specific combination—one that is not currently available on the market, and which may never even have existed—and to establish it as

To breed for 'purity' of breed. One can therefore, with a species that has been sufficiently analysed to begin with, proceed in much the same way as a chemist, synthesising and quite deliberately produce specific, desired combinations of properties

i.e. to create specific new breeds.

What we perceive as a single trait in a fully developed organism—such as a particular coat colour—is, as we have already seen from a whole series of examples, always determined by a large number of hereditary factors; one need only think, for example, of the many factors that play a part in the development of the wild colour in rabbits. It is also frequently the case that completely different and mutually independent factors manifest themselves in exactly the same way . A schematic example probably illustrates this most clearly: the fact that a wheat variety is fairly immune to a parasitic fungus may be due to the fact that, because of the structure of its cell walls, it is particularly well protected against the penetration of the fungus's haustoria.

Another variety of wheat may exhibit exactly the same degree of immunity, but in this case the immunity is based, for example, on the presence of certain protective substances in the cell sap. If one crosses the two varieties, which are equally immune and perhaps also outwardly indist , one will obtain a hybrid that has now inherited both causes of immunity, but carries each only in a heterozygous state and is therefore — provided there is only partial dominance — is likewise only approximately as immune as either of the two parent races. In the F_2generation of these hybrids, however, there must also appear plants that are homozygous, having inherited **both** causes of immunity, and are therefore twice as immune as the parent races were; furthermore, there must appear — in addition to the wholly recessive combinations — plants that have inherited n o e i t h e r of the two causes, and are consequently n o t i m m u n e a t a l l; and finally, a range of intermediate types must arise, whose immunity corresponds approximately to that of the parent races. A very instructive case of this kind, described by Nilsson-Ehle—to whom we owe the elucidation of these initially incomprehensible patterns of inheritance—shall be discussed in more detail. It is likely that similar circumstances are very common in humans. In wheat, red grain colour is determined by at least three factors—**R**, **S** and **T**. A plant that is heterozygous for only one of these factors—for example, **Rrsstt** or **rrSstt**, etc.—is very pale red; a plant that is homozygous for one of these factors—for example, **RRsstt** or **rrSStt**, etc.—is a slightly darker red; and

finally, at the other end of the spectrum, plants that are homozygous for all three factors — **RR SS** TT — are the darkest red.

If one crosses a white variety that contains none of these factors (**rr ss tt**) with a homozygous dark-red one (**RR SS TT**), one obtains a hybrid **Rr Ss Tt**, which occupies an intermediate position in colour, being pale red in colour. This hybrid produces eight gametes: **RST**, **RSt**, **RsT**, **Rst**, **rST**, **rsT**, **rst**; and these eight types of gamete can combine in the ways shown in the table opposite.

The second column specifies the grain colour of the individual categories; the darkest red, determined by the three homozygous factors, is designated as red VI, white as red 0, and the lightest red, determined by only one heterozygous factor, as red I; the remaining colour gradations are designated accordingly.

The table shows that in the F_2generation of the cross, alongside very rare plants which display exactly the same colouring as the parental lines, a long series of differently coloured intermediate stages must occur, namely plants coloured more or less dark red with colour gradations ranging from red I to red V.

Possible combinations of gametes	F_2		F_3
1) RST × RST	red	VI	red [B]
2) RST × RSt	"	V	"
3) RST × RsT	"	V	"
4) RST × Rst	"	IV	"
5) RST × rST	"	V	"
6) RST × rSt	"	IV	"
7) RST × rsT	"	IV	"
8) RST × rst	"	III	divided into 63 red : 1 white
9) RSt × RST	"	V	red
10) RSt × RSt	"	IV	"
11) RSt × RsT	"	IV	"
12) RSt × Rst	"	III	"
13) RSt × rST	"	IV	"
14) RSt × rSt	"	III	"
15) RSt × rsT	"	III	divided into 63 red : 1 white

[16]) RSt × rst	"	II	"	" 15 "	: 1 "
[17]) RsT × RST	"	V	red		
[18]) RsT × RSt	"	IV	"		
[19]) RsT × RsT	"	IV	"		
[20]) RsT × Rst	"	III	"		
[21]) RsT × rST	"	IV	"		
[22]) RsT × rSt	"	III	divided into 63 red		: 1 white
[23]) RsT × rsT	"	III	constant		
[24]) RsT × rst	"	II	divided into 15 red		: 1 white
[25]) Rst × RST	"	IV	red		
[26]) Rst × RSt	"	III	"		
[27]) Rst × RsT	"	III	"		
[28]) Rst × Rst	"	II	"		
[29]) Rst × rST	"	III	divided into 63 red		: 1 white
[30]) Rst × rSt	"	II	"	" 15 "	: 1 "
[31]) Rst × rsT	"	II	"	" 15 "	: 1 "
[32]) Rst × rst	"	I	"	" 3 "	: 1 "
[33]) rST × RST	"	V	red		
[34]) rST × RSt	"	IV	"		
[35]) rST × RsT	"	IV	"		
[36]) rST × Rst	"	III	splitting into 63 red		: 1 white
[37]) rST × rST	"	IV	red		
[38]) rST × rSt	"	III	"		
[39]) rST × rsT	"	III	"		
[40]) rST × rst	"	II	split into 15 red		: 1 white
[41]) rSt × RST	"	IV	red		
[42]) rSt × RSt	"	III	"		
[43]) rSt × RsT	"	III	divided into 63 red		: 1 white
[44]) rSt × Rst	"	II	"	" 15 "	: 1 "
[45]) rSt × rST	"	III	red		
[46]) rSt × rSt	"	II	"		
[47]) rSt × rsT	"	II	divided into 15 red		: 1 white
[48]) rSt × rst	"	I	"	" 3 "	: 1 "
[49]) rsT × RST	"	IV	red		
[50]) rsT × RSt	"	III	divided into 63 red		: 1 white
[51]) rsT × RsT	"	III	red		
[52]) rsT × Rst	"	II	per fess in 15 red		: 1 white

53) rsT × rST	"	III	divide	
54) rsT × rSt	"	II	d into 15 red	: 1 white
55) rsT × rsT	"	II	red,	
56) rsT × rst	"	I	divided into 3 red	: 1 white
57) rst × RST	"	III	divided into 63 red	: 1 white
58) rst × RSt	"	II	" " 15 "	: 1 "
59) rst × RsT	"	II	" " 15 "	: 1 "
60) rst × Rst	"	I	" " 3 "	: 1 "
61) rst × rST	"	II	" " 15 "	: 1 "
62) rst × rSt	"	I	" " 3 "	: 1 "
63) rst × rsT	"	I	" " 3 "	: 1 "
64) rst × rst	white	0	whit e	

If we examine the table to see how frequently each colouring must appear in F_2, a count yields the following:

Of the 64 possible combinations, there are:

1	combination	plants with the colour red				VI	(such as the one P1 plant)
6	combinations	"	"	"	"	V	
15	"	"	"	"	"	IV	
20	"	"	"	"	"	III	
15	"	"	"	"	"	II	
6	"	"	"	"	"	I	
1	Combination	"	"	"	"	0	(white, like the other P_1plant)

It is therefore to be expected that $F_2$from such a cross will consist of a very large number of plants exhibiting a moderate intensity of red colouration, and of very few which are very dark, as well as a very small number which are very light. Furthermore, a glance at the sequence of numbers 1, 6, 15, 20, 15, 6, 1,
which indicates the frequency of the individual intensities, such that this sequence of numbers corresponds to **the random curve**.

Fig. 9.
Wheat ear shapes.
a) Loose-spiked land wheat,
b) Square-head wheat,
c) Compactum wheat.

Thus, if, as in our example, the grain colour is influenced by a number of factors that are independently inherited and also express themselves independently of one another, but whose effects add up, then a plant that is heterozygous for several of these factors

will produce offspring exhibiting a whole range of colour shades; plants with a medium intensity of colour will be very common, whereas very light and very dark plants will be rarer—and, in accordance with the random curve, will become increasingly rare the lighter or darker they are.

It is likely that most cases of hybridisation can be understood in this way; in the literature, it is stated that the hybrids occupy an intermediate position between their parents and do not segregate, but instead consistently pass on this intermediate position. For example, regarding the inheritance of skin colour in a 'Negro × European' cross, it is claimed that the resulting mulattoes would produce offspring whose skin colour remains constant. However, according to careful recent studies, this is not the case. The marriage of two F_1mulattoes may — albeit only very rarely — produce children with white or black skin, but the vast majority of children from such marriages have skin colour approximately the same as that of the F_1mulattoes. If one assumes that the difference in skin colour between Black people and Europeans — much like the grain colour in the wheat example discussed above — is determined by several independently Mendelian co-dominant factors, then the finding is: 'very isolated cases of children resembling the parent races, the vast majority of children being approximately the same as the F_1 -Mulattoes' is readily understandable.

Factors acting in the same direction are particularly common when one investigates the inheritance of physical characteristics and the like. The peculiar patterns of inheritance that can arise here will also be briefly illustrated using a breeding experiment with wheat. The length of a wheat ear is influenced by a whole range of hereditary factors. Firstly, we know of at least two factors acting in the same direction which make the ears long and loose; let us call these two factors **L** and **M**. A plant with the genotype **LLMM** has very long and loose ears (Fig. 9a); all plants with one of these factors, **llMM** or **LLmm**, are medium-length-eared wheat; and finally, an llmm plant is a
'squarehead' (Fig. 9b). A further key factor, **C,** makes the ears very short and compact (Fig. 9d). **C** is dominant over **L** and **M**; thus, plants with the genotypes **CC LL MM**, **CC ll MM**, **CC ll mm**, **Cc LL MM**, etc., are all externally indistinguishable 'compactum'

wheat. Based on this finding, a great many of the peculiar crossing results described in * * become perfectly understandable. For example, under certain circumstances, the crossing of two medium-spiked varieties may produce a hybrid that is also medium-spiked; however, in the F_2generation, alongside many medium-spiked plants, very long-spiked and loose-spiked varieties, as well as very short-spiked ('Squarehead') wheat, now appear. The cross would then have followed this pattern:

c c L L **m m**	×	**c c l l** **M M**	P_1
medium length		medium length	
c c L l M m			$F1$
medium-length			

This should result in F_2producing, amongst others, **cc LL MM** — (long, loose-spiked) and **cc ll mm** — (Squarehead) plants.

Furthermore, the cross **CC LL MM** (compactum) × **cc ll mm** (Squarehead) must produce only compactum plants (CcLlMm) in $F_{(1)}$and in F_2there is then a split into compactum, very long and loose-spiked, medium-length and Squarehead.

When conducting research into the laws of inheritance of racial differences in humans—for example, the mode of inheritance of skull shape and the like—one must constantly bear in mind these clear and transparent examples from the plant kingdom. The case where, in the cross between two races, F_1 shows, more or less clearly, a dominance of one parent, and where in F_2 a neat and very simple segregation occurs—that is, the textbook case of Mendelian inheritance—is rarely found. One must always bear this in mind. One must not imagine that one is qualified to speak authoritatively on heredity merely because one has grasped the very simplest laws of segregation. Unfortunately, a great many people still labour under this misconception.

Factorlinkage.

If one carries out cross-breeding experiments, such as those mentioned above, with any organism, sooner or later one invariably encounters a phenomenon known as factor linkage.

Let us again take a simple example: the small fruit fly *Drosophila ampelophila* is today by far the animal whose genetic factors have been most thoroughly investigated. Around 100 genetic factors have already been clearly identified. However, for the time being, we shall consider only two of these, namely the two factors **Y** and **W**. A strain possessing these two factors, as well as all other factors for normal body structure, looks like an ordinary wild Drosophila. A strain with the same genotype but **y y** has a yellow body colour, and a strain with this genotype but **w w** has white eyes. If one now crosses a lineage with the genotype **Y Y W W**,
i.e. a dark-coloured Drosophila, with a strain of the genotype **y y w w**, i.e. a yellow individual with white eyes, one obtains a hybrid **Y y W w**, which has dark body and eye colour.

According to what we have heard, this hybrid should produce its four types of gametes – **Y W**, **Y w**, **y W**, **y w** – in equal frequencies, and in $F_2$the four types of individuals – dark with dark eyes, dark with white eyes, yellow with dark eyes, and yellow with white eyes – should occur in a ratio of 9: 3: 3: 1; or, if such hybrids are back-crossed with the fully recessive strain (yellow with white eyes), i.e. **Y y W w** × **y y w w**, then, according to what we heard on p. 26, one would expect the four types of individuals to occur in a ratio of 1: 1: 1: 1. However, neither of these is the case.

In the typical F_2generation, one does not obtain the ratio 9: 3: 3: 1, but rather dark dark-eyed and yellow white-eyed animals are present in relatively far too many, whilst dark white-eyed and yellow dark-eyed animals are far too few . In the backcross, one does indeed obtain the four types of animals, but dark white-eyed and yellow dark-eyed animals are far fewer in number than the

other two types. All this is due to the fact that the hybrid **Y y W w** does not produce its four types of gametes in equal numbers, but rather in unequal proportions; specifically, in this case, the gametes are found in approximately the following proportions:

99 **Y W** : 1 **Y w** : 1 **y W** : 99 **y w**.

A hybrid of the same formula, **Y y W w**, can also be produced in a second way, namely by the cross **Y w** × **y W**. This hybrid then produces its gametes in the ratio 1 **Y W** : 99 **Y w** : 99 **y W** : 1 **y w** and exhibits the corresponding frequencies of the four types of individuals in $F_2$and in the backcross. It can therefore be said that two factors have a marked tendency to remain together, just as they were already together in the P_1animals , that they are, so to speak, 'linked' in a sense, and that this linkage was interrupted in only a small percentage of cases during gamete formation.

It is very common for two genetic factors to be is very common; this matter is also of great interest in human inheritance and is of the very greatest theoretical importance for our entire conception of the cytological basis of the entire Mendelian segregation and the racial differences in general.

Fig. 10.
Chromosomes (of diploid cells) of a bug (*Anasa)*: top, in their natural arrangement; bottom, arranged in pairs side by side.
(After Wilson).

We must therefore address this question very briefly here as well. We had previously discussed that every hereditary racial difference must ultimately be based on some difference in the chemical composition or the finer structure of the idioplasm.

Furthermore, we can now say with complete certainty that, for the most part, the idioplasm — for the time being a purely theoretical concept derived from genetics — must be localised in the cell nucleus, and the research of recent years also makes it highly probable that one of the differences can be attributed to a difference between

two homologous chromosomes. It may well be assumed here that, in all sexually reproducing organisms, each gamete possesses a single 'haploid' 'set' of chromosomes (, Drosophila 4; in the frog, Rana fusa 12, in the guinea pig 8, and so on), and the fertilised egg cell resulting from the union of two gametes has a double 'diploid' set of chromosomes. Thus, in Drosophila there are 4, in the frog 12, in the guinea pig 8, etc., pairs of chromosomes. The individual chromosomes often have different shapes, so that in diploid cells the individual pairs of chromosomes belonging together can be recognised quite clearly (see Fig. 10).

At some point, the diploid cells undergo a
'reduction division', cells are formed, each containing only a single, haploid set of chromosomes, with the paternal and maternal chromosomes being shuffled. The cells thus formed can — as is the case with all higher animals — serve directly as gametes, or — as is particularly the case with most plants — only later give rise to gametes following a further series of divisions, the number of which varies but which do not alter the chromosome complement. This distribution of chromosomes during reduction division could well form the basis of the entire Mendelian segregation. Let this be briefly illustrated by a schematic example.

Let us assume that we have a living organism with — haploid — three chromosomes; one lineage of this organism has chromosomes **A**, **B** and **C**, whilst another has chromosomes **a**, **b** and **c**. We further assume that these chromosomal differences A—a, B—b, C—c are the causes of three independently Mendelian fundamental differences between these two lineages.

All the diploid cells of the hybrid of these two lineages would then have the diploid chromosome set **A a B b C c**. During the meiotic division of such a hybrid, cells are once again produced, each containing only one haploid chromosome set. Whether this set consists entirely of paternal or entirely of maternal chromosomes, or whether the various conceivable mixed sets arise, is equally probable. The following eight haploid chromosome sets are possible: ABC, ABc, AbC, Abc, aBC, aBc, abC, abc, and we

may expect that, out of a large number of cells, one in eight will contain one of these chromosome sets. In other words, such a hybrid produces eight different types of gametes with equal frequency. This line of reasoning thus shows that precisely the eight types of gametes must arise which, according to Mendel's theory, a hybrid of the form l **A a B b C c** actually produces.

One could therefore very well imagine that the distribution of hereditary factors amongst the gametes of a hybrid has its physical basis in these cytological processes.

Fig. 11.
Schematic representation of chromomere exchange between two homologous chromosomes. a, a_1, a_2 Formation of mixed chromosomes (a_2) through a single break in the chromomere chain (single crossing-over, as described by the American authors). b, b_1 Formation of mixed chromosomes through a double exchange (double crossing-over, as described by the American authors).

According to the latest research, this is certainly the case, albeit with one very significant caveat.

The very fact that, in some well-studied organisms, far more Mendelian hereditary factors are known than there are pairs of chromosomes suggests that the situation must be somewhat different.

The individual chromosomes themselves consist in turn of segments, which we shall call chromomeres. Even before meiosis, there is probably a kind of exchange of segments—that is, of more or longer long segments of the chromomer chain. This is best visualised as the two chromosomes tearing apart at any random point and the resulting segments being exchanged. In this way, chromosomes are formed that are composed of paternal and maternal segments (cf. Fig. 11).

It is now highly probable that the cytoplasmic basis of a racial difference, which is treated as a single unit, is to be sought in a difference in the structure or chemistry of two homologous chromosomes.

The fact that, during the formation of gametes in a hybrid that is heterozygous for *n* factors, as many different types of cells are formed as there are possible combinations between the *n* factors, is explained in this model by the fact that, firstly, the two sets of chromosomes, as discussed above (p. 47), are distributed completely at random during reduction division — that is, in every possible combination of paternal and maternal chromosomes — and that, secondly, segments of two homologous chromosomes are also exchanged with one another. Thus, in a sense, a double mechanism ensures the distribution of both types of genetic material and the achievement of all possible combinations. However, whilst the individual **chromosomes** of a set are generally not connected to one another and are distributed to the daughter cells during meiosis completely freely and independently of one another, the individual **chromomeres** of a chromosome are more or less firmly bound together. A chromosome does not, for example, break down into all its chromomeres, which are then freely exchanged with the chromomeres of the homologous chromosome; rather, only larger or smaller segments—that is, entire series of chromomeres—are exchanged.

This must mean that two Mendelian factors, which are located on two different chromosomes, segregate freely and independently of one another, i.e. that the four gametes, which represent the four possible combinations, are produced in equal numbers; but that two Mendelian factors, which are based on differences in the structure of two pairs of chromomeres of the same chromosome, do not segregate independently, but rather that, of the four possible gametes, which occur in greater numbers, in which the two hereditary factors are present in that particular combination, in which they were introduced into the cross. In other words**, two genetic factors located on the same chromosome must exhibit the phenomenon of factor linkage described earlier** **on p. 44**.

In itself, the chromomer exchange alone would suffice to explain the free Mendelian segregation and the phenomena of linkage. Even if only this chromomer exchange were to take place, and not an exchange of the entire chromosomes as well, hereditary factors localised in two different chromosomes would still be expected to segregate freely, whilst those localised on the same chromosome would be expected to show linkage. However, many cytological observations suggest that, in addition to the chromomer exchange—which presumably takes place in the early stages of nascent nuclear division—the exchange of entire chromosomes occurs at a later stage; that is to say, as already mentioned above, a dual mechanism ensures the distribution of individual variations.

From this entire theoretical framework, further conclusions can now be drawn which have been confirmed in the most thorough manner by heredity experiments, thereby greatly reinforcing this whole theory of the cytological basis of Mendelian segregation. The first conclusion is that, if the theory is correct, then every organism must possess so many groups of hereditary factors that are more or less strongly linked to one another, than the number of haploid chromosomes that this organism possesses. This conclusion holds true for Drosophila — the only species to have been studied with sufficient care in this regard to date. For Drosophila, thanks to the pioneering work of the Morgan school, we are currently aware of around 100 Mendelian genetic factors, which fall into four groups of factors that each exhibit factor linkage amongst themselves. However, as we heard earlier, Drosophila does indeed have four chromosomes.

The second conclusion is: if the chromomeres of a chromosome are linked in a chain-like manner, and if, prior to meiosis, whole sections of the chain are exchanged between two homologous chromosomes, then two chromomeres situated at the two opposite ends of a chromosome are separated from one another by each such chromosome break and subsequently distributed to two different daughter cells; and the closer two chromomeres are to one another within a chromosome, the lower the probability that the break will occur precisely between

them, i.e. there must be gradations in the degree of linkage; individual genetic factors within a
'linkage group' must be strongly linked, whilst others are weakly linked; moreover, the strength of linkage must always be the same for a given pair. This, too, undoubtedly holds true for the organisms that have been sufficiently studied to date — only Drosophila and Antirrhinum are seriously under consideration. In the case of Drosophila, which is the only species to have been studied to the necessary extent so far, it is even possible today to produce topographical maps of the individual chromosomes, showing how the genetic factors must be arranged within them. Let this be illustrated by an example: a hybrid **A a B b**, which is heterozygous for two factors **A** and **B**, produces—in the absence of linkage, i.e. when the two factors are located on different chromosomes—the four gametes **A B**, **A b**, **a B** and **a b** in equal numbers. If, on the other hand, the factors are located on the same chromosome – that is, if there is linkage – the gametes are produced in the ratio *n*: 1: 1: *n* or 1: *n*: *n*: 1, depending on whether the hybrid was produced via the pathway **A B** × **a b** or via the pathway **A b**
× **a B**.

Depending on the strength of the linkage, the ratio 1: *n* is a varying number, but always less than 1. In order to obtain comparable figures, we shall continue to express this value 1: *n* as a decimal fraction.

In *Drosophila*, a number of factors—which we shall designate as **Y**, **W**, **O**, **M** and **R**, and whose modes of action are set out in the table below—exhibit a more or less strong coupling amongst themselves.

Factor	Mode of action
Y	**Y** is a prerequisite for the development of dark body colour; all yy individuals have a yellow body colour.
W	**W** is a prerequisite for dark eye colour; all **ww** individuals have white eyes.
V	**V** is also a prerequisite for dark eye colour; all vv animals have the colouration described by Morgan as 'vermilion' (a scarlet-like red).
M	**M** is a prerequisite for properly formed wings; all **mm** animals have short 'miniature' wings.

R	**R** is also a prerequisite for properly formed wings; all **rr** individuals have stunted 'rudimentary' wings.

The varying degrees of linkage found for these five factors in Morgan's experiments are, in turn, presented in tabular form below.

1	2	3
Pair of factors	shows the empirical value for 1 : n in the experiments	this value, expressed as a decimal fraction, is
Y W	354/32218	0.011
Y V	2117/6221	0.340
Y M	1054/3063	0.344
Y R	605/1420	0.426
W V	4336/13395	0.324
W M	7591/22910	0.331
W R	894/2136	0.419
V M	50/1640	0.030
V R	183/850	0.215
M R	1562/9295	0.168

If one assumes the value of the ratio 1:*n*, found for the linkage of two factors, to represent the extent of their distances on the chromosome, it follows initially that **Y—R** must be furthest apart and that the remaining factors must be arranged between them in the manner shown in Fig. 12.

Fig. 12. Spatial distribution of a number of hereditary factors on a chromosome of Drosophila.

It naturally follows from this entire theoretical framework that, if, for example, the linkage coefficient for **Y—W**—i.e. the theoretical distance—is taken to be 0.011, and the linkage coefficient between **W** and **M**—i.e. in the Theory has found the distance to be 0.331, so that for **Y—M** the linkage coefficient (distance) is found to be 0.011 + 0.331. If this regularity were not generally confirmed, i.e. if the

calculation of the position of a factor from its various linkages did not always yield the same point on the chromosome, conceived as a line segment, then the whole theory might be incorrect. However, the fact that this very assumption always holds true provides the theory with its current firm foundation. A glance at the table on p. 52 shows that the linkage coefficient of 0.344 found for the **Y–M** factors is in fact approximately equal to 0.331
+ 0.011, and that this conclusion is generally satisfied to a close approximation in other respects as well. We consistently find, however, that the coupling coefficient found directly for two factors (e.g. the figure of 0.426 found for **Y** and **R**) is always slightly smaller than the figure calculated for the same factors from their couplings with intermediate factors (for **Y** and **R**, as a glance at Fig. 12 shows, the calculated figure is 0.011 + 0.324 + 0.030 + 0.168 = 0.533). However, the fact that the directly observed linkage coefficient is slightly smaller than the calculated value follows from the theory itself; specifically, the greater the linkage coefficient is in absolute terms, the greater the difference between the observed and calculated values must be, i.e. the further apart the two factors are within the chromosome. This is a consequence of the fact that, when a double exchange of segments takes place in a pair of chromosomes — according to the diagram in Fig. 11b — the chromomeres that are far apart end up being located together again within the same chromosome.

Naturally, one can also draw a conclusion, in reverse, from the magnitude of the difference between the observed and calculated linkage coefficient regarding the frequency with which such a double exchange occurs in the species in question.

Linking of factors is not merely partial, as in the cases discussed so far, but there is also complete linkage,
that is to say, two different factors do not segregate freely from one another at all, but always together. Such cases of absolute linkage of several factors (usually referred to in American and English literature as ‘multiple allelicism’) have been found in all thoroughly studied organisms. Apart from cases of complete linkage with the sex factor (p. 54), they are most likely due in the majority of cases to the fact that the factors in question are based on

several differences in the finer structure of one and the same pair of chromomeres.

The whole phenomenon of factor linkage is, as mentioned, very widespread; if we have so far identified only very few certain cases in humans, this is probably due solely to the fact that humans have so far been studied in a highly inadequate manner.

Theinheritance of sex.

The study of factor linkage has, amongst other things, gained great importance for the question of the mode of inheritance of sex. There is no longer any doubt today that, in organisms with two sexes—that is, with males and females—the sexual difference itself is inherited according to the laws of segregation. One sex is always homozygous, the other heterozygous.

For example, in the fruit fly *Drosophila*, the sexual dimorphism is determined by a genetic factor which we shall call **X**. All females are **X X**, and all males are **X x**. Every mating is therefore a backcross according to the following pattern:

X X × **X x** P_1

, 50% **X X** 50% **X x** F_1

.

This simple assumption readily explains the fact that, in every reproduction, males and females are produced in roughly equal proportions. That the assumption holds true is clearly demonstrated by the fact that there are linkages between this factor **X** and the other factors located on the same chromosome. These linkages between other hereditary factors and the sex-determining factor give rise to a series of very peculiar patterns of inheritance, which are referred to as sex-linked inheritance or, to use a less apt term, sex-restricted inheritance. This will be illustrated by a few examples. As mentioned earlier, there is, amongst others, a strain of Drosophila that has white eyes, whilst the wild-type strain has dark (red) eyes. Red eyes are dominant over white eyes. Here, too, let us denote the factor responsible for this difference by **W**; **ww** is therefore a white-eyed individual, **WW** is homozygous, and **Ww** is heterozygous for red eyes. This factor **W** is completely, or as is usually said, **absolutely** linked to the sex factor **X**.

If a normal red-eyed female is crossed with a white-eyed male, the F_1 generation consists of red-eyed males and red-eyed females. The F_2 generation, obtained by interbreeding the F_1 individuals, consists of red-eyed females and red-eyed and white-eyed males.

Almost exactly half of the males are red-eyed, the other half white-eyed. The white-eyed grandfather has therefore passed on his eye colour to half of his **grandsons**, but to **none of his granddaughters**. As can be seen from the diagram below, this pattern of inheritance must result from the linkage.

P_1. red-eyed female **XX WW** × white-eyed male **Xx ww**

Gametes of P_1: all **XW** | 50% **Xw**, 50% **xw**

F_1. 50% each of **XX** and **Ww**, and **Xx Ww**
(red-eyed females; red-eyed males)

Gametes from F_1: 50% **XW** and 50% **Xw** | 50% each of **XW** and **xw**

Possible combinations of these F_1 gametes i.e. different types of F_1 organisms:

XW × XW = XX WW = red-eyed females
XW × xw = Xx Ww = " males **Xw × XW**
= XX Ww = " females **Xw × xw = Xx xw**
= white-eyed males

The peculiar pattern of inheritance of eye colour is therefore easily understood on the basis of linkage.

In F_2 of this cross, no white-eyed females appear, but it is possible to obtain white-eyed females by other means. If one crosses a number of the red-eyed females obtained in F_2 of the cross just discussed with white-eyed males, then in half of these matings, red-eyed and white-eyed females, and red-eyed and white-eyed males, will be produced in equal proportions. The reason for this is as follows. Half of the F_2 females from the first experiment discussed, as can be seen from the diagram, have the genotype **XX Ww**, and when

of these animals with the **Xx ww** males must yield the following combinations:

XW × **Xw** = **XX Ww** = red-eyed females, **XW** × **xw** = **Xx Ww** = ‘ males, **Xw** × **Xw** = **XX ww** = white-eyed females, **Xw** × **xw** = **Xx xw** = ‘ males.

It follows from these theoretical considerations that both types of male and both types of female must be produced in equal proportions, exactly as was found in the experiment. This linkage of individual genetic factors with the sex factor now reveals a further peculiarity. Whilst chromomeric exchange normally takes place between two homologous chromosomes, it always fails to occur between two homologous chromosomes which differ in the sex factor. This is probably easiest to demonstrate once again using a Drosophila example. We cross two Drosophila strains which, apart from a factor determining eye colour, also differ in a factor determining body colour: namely, a grey, red-eyed **YY WW** strain with a yellow, white-eyed **yy ww** strain. In Drosophila, the **Y** factor, which determines the grey body colour, is also located on the same chromosome as the sex factor **X** and the eye factor **W**. The cross between a grey, red-eyed female and a yellow, white-eyed male proceeds according to the following scheme:

P_1.	grey, red-eyed female **XX YY WW**	×	yellow, white-eyed male **Xx yy ww**
Gametes	all **XYW**		50% **Xyw**, 50% **xyw**
F_1.	50% **XX Yy Ww** i.e. grey, red-eyed females	and	50% **Xx Yy Ww** i.e. grey, red-eyed males

The peculiarity mentioned earlier is now evident in the formation of gametes in these F_1 animals. In the females, the two homologous chromosomes do not differ in the sex factor. Consequently, a chromomer exchange takes place here, and the females therefore produce the four possible categories of gametes, i.e.

the egg cells **XYW**, **XYw**, **XyW** and **Xyw**, but, as might be expected, not in equal numbers; rather, they occur in the ratio 84 : 1 : 1 : 84, i.e. the two types of cells (**XYw** and **XyW**), which can only arise through chromomer exchange, are found in smaller numbers than the others. The formation of gametes in males proceeds quite differently. Here, according to what has been said previously, no exchange of chromomerena takes place at all, because the homologous chromosomes in which the factors **X**, **Y** and W are located, are also different in terms of sex factor; one chromosome is **XYW**, the other **xyw**. Consequently, these **Xx Yy** Ww males produce only two types of spermatozoa: **XYW** and **xyw**. During fertilisation of the F_1 animals among themselves, the following combinations are possible between the four different egg cells and the two different sperm cells:

XYW × **XYW** = **XX YY WW** = grey, red-eyed females
XYW × **xyw** = **Xx Yy Ww** = " " males **XYw** ×
XYW = **XX YY Ww** = " " females **XYw**
× **xyw** = **Xx Yy ww** = " white-eyed males **XyW** × **XYW** =
XX Yy WW = " red-eyed females **XyW** × **xyw** = **Xx yy**
WW = yellow " males **Xyw** × **XYW** = **XX Yy Ww** = grey
„ females **Xyw** × **xyw** = **Xx yy ww** = yellow, white-eyed
males

Thus, a l l f e m a l e s a r e grey-eyed and red-eyed; amongst the males, however, a l l f o u r t y p e s o f a n i m a l s are present: grey-red, grey-white, yellow-red and yellow-white. However, because the four types of egg cells from which the four types of males arise are not produced with the same frequency, but in the ratio **84 XYW : 1 XYw : 1 XyW : 84 Xyw,** the four types of males also occur in the ratio of **84** grey-red: 1 grey-white: 1 yellow-red: 84 yellow-white. In total, therefore, such an F_2generation consists of:

170 grey, red-eyed females (6080)
84 " " males (2870)
1 " white-eyed " (36)
1 yellow, red-eyed " (34)
84 " white-eyed " (2373)

The figures in brackets indicate the frequency with which the various categories of animals were found in a specific experiment (Morgan). The figures demonstrate the extent to which theory and experimental results coincide in this instance. However, they also illustrate the immense amount of work required to clarify these relationships.

The study of the linkage of other factors with the sex factor continues to be of particular interest due to the fact that it is often possible to distinguish **X chromosomes** from x chromosomes under a microscope. For example, in Drosophila, the X chromosomes are slightly longer than the x chromosomes. Accordingly, female Drosophila have four pairs of chromosomes in their somatic cells, each pair consisting of two partners of equal length. In males, on the other hand, one pair — namely the chromosomes in which the sex difference lies — consists of two unequal partners. In some animals, the differences in size are very striking, and differences in shape are also present. In many cases, we also find that the X chromosomes are entirely absent; in these animals, the eggs contain *n* chromosomes, whilst the spermatozoa contain partly *n* and partly n–1 chromosomes. The somatic cells of females have *2n* chromosomes, whilst those of males have 2n–1 chromosomes.

There is now little doubt that sex determination in dioecious organisms generally occurs in the manner just described. It is striking that in some organisms the female sex is heterozygous, whilst in others, such as D r o s o p h i l a, the male sex is heterozygous. In some cases, this can already be recognised microscopically by the difference in the chromosomes; in most cases, however, it can only be deduced from the pattern of inheritance in sex-linked inheritance.

In humans, as in D r o s o p h i l a, the male sex is heterozygous.

We are already familiar with a large number of cases of sex-linked inheritance in humans. (More on this later.)

Apart from the linkage of certain hereditary factors to the sex factor, sex-linked inheritance can also give rise to other complications. For example, the dominance of a trait may differ between the two

sexes, or a particular genetic factor may be expressed in only one sex whilst remaining inactive in the other, which then results in, for example, two distinct races of females being distinguishable, whilst the corresponding males are not outwardly different, yet the females nevertheless pass on the racial characteristics of their respective races. However, it would be going too far to discuss all such cases here; reference must be made to the relevant specialist literature.

The fact that sex is inherited according to the laws of segregation also implies that it is determined for each individual in the context of fertilisation. In humans, all oocytes are genetically neutral; of the spermatozoa, one half carries the genetic predisposition for male, the other half that for female. Accordingly, one would normally expect the sex ratio to be 1:1. However, it is well known that the ratio of female to male births is by no means 1:1, but, for example, in Germany
100 : 105.2. If one considers not only the ratio of live-born children, but also takes into account all stillbirths and all abortions where the sex can already be determined, one even arrives at a ratio of 100 female births to almost 150 male births. We do not know the cause of this shift in the sex ratio. It may, for example, be due to the fact that, of the two types of spermatozoa produced by every man, the male-determining ones are better suited to travelling the long distance from the vagina to the oviduct; in other words, a kind of selection takes place here. A thorough experimental investigation of this question would also be of the utmost practical importance, because, given everything we know today about the inheritance of sex, this is the only area in humans where possibilities exist to alter the sex ratio at will, that is, to influence the sex of a child to be conceived with a certain degree of probability.

According to the current state of knowledge, it seems impossible to arbitrarily determine the sex of a human embryo even after fertilisation. XX embryos are and remain female in humans; XX embryos are and remain male. In

other organisms — frogs, for example — the situation is, by contrast, quite different. For this question too, reference must be made to the specialist literature.

Even partial, let alone absolute, factor linkage shows us that Mendel's laws of segregation do not apply without restriction to all racial differences. Only those racial differences in accordance the laws of segregation, whose developmental mechanism consists of only minor differences in the structure of homologous chromomeres. Mendelian segregation also occurs only where the entire complex apparatus of chromomer exchange, chromosome distribution, etc.—that is, the entire mechanism of reduction division—functions without disruption. However, as soon as breeds are crossed which exhibit such great differences in nuclear structure that this mechanism is in some way disrupted, Mendelian segregation is also disrupted. We therefore find that, although highly distinct species can often still be crossed and even produce fertile hybrids, but further inheritance proceeds according to completely different laws, which vary from case to case.

It is, however, frequently found that closely related species, even if morphologically very different, still show quite regular back-breeding when crossed, whilst in the F_2generation, they give rise to a truly immense variety of new combinatorial types, but the proportion of hybrids is different.

However, a discussion of the whole question of interspecific hybridisation can be omitted at this point, simply because, as far as we know, when different human races interbreed, inheritance essentially follows the laws of segregation. Nothing is known about crossbreeding between humans and distantly related species, such as the closest-related great apes, and it is also highly unlikely that such a cross would produce a viable offspring.

On the basis of the theoretical concepts presented here regarding the cytological cause of Mendelian segregation, inheritance according to laws other than Mendel's is also to be expected for all racial differences that are not rooted in the chromosomes but somewhere else in the idioplasm. Differences in the structure of the rest of the filamentous framework of the cell nucleus, in the structure of the centrosomes, etc., must surely also entail some differences in the resulting organisms. Now, however, we are aware of individual racial differences in many plants that most certainly do not follow Mendelian inheritance, even though all other differences between the races in question are subject to the laws of segregation. We also have certain indications that, for example, some such non-Mendelian variations must be due to differences in the structure of the chromatophores (pigment-bearing cells). But on the whole, so little is known for certain about this today that a detailed discussion of this question, too, can probably be omitted. This is all the more feasible given that we know absolutely nothing about racial differences in humans that are certainly non-Mendelian. It is surely sufficient to bear in mind at all times that the Mendelian laws of segregation do not apply without exception, and that, sooner or later, also in humans encounter instances of inheritance that do not conform to these laws.

Let us briefly summarise once more what has been discussed regarding mixovariation: a second main reason why the children of a pair of parents differ from their parents and also from one another is that, in sexual reproduction, there are usually two different idioplasms, hereditary tendencies—or whatever else one might call them—and that, during the formation of the gametes of the hybrids, these different hereditary tendencies are distributed amongst the individual cells according to very complex laws and rules. In the offspring of two crossed individuals, all possible new combinations of the original differences arise.

For the vast majority of racial differences, recombination follows the rules discovered by Gregor Mendel, which, admittedly, have been elaborated in great detail

. However, certain racial differences and a great many species differences are inherited and combined according to other laws, some of which are very little known.

The continuous kaleidoscopic arising and disappearance of new combinations of a certain number of original races is the main cause of the hereditary variation in both self sexually reproducing organisms. This also applies in particular to human beings.

c) Idiovariations.

As we heard in the previous chapter, the ever-changing combinations of a certain number of fundamental differences give rise to an immense potential for variation, and several biologists have in all seriousness put forward the view that, in fact, a l l hereditary variations are essentially mixovariations. But that is certainly not the case. There are large classes of organisms—such as bacteria, blue-green algae (Cyanophyceae), many algae and a great many filamentous fungi—which reproduce exclusively asexually, and yet the emergence of new, hereditably distinct races occurs here too. Quite apart from that, however, there can be no doubt whatsoever today that new hereditary differences are constantly arising before our very eyes, even among organisms that reproduce sexually.

Whether any individual that is genetically distinct from its parents and siblings is merely a new combination—perhaps resulting from linkage or other causes—of pre-existing fundamental differences that occur only rarely, whether there is perhaps an irregular division, or whether a new, previously non-existent fundamental difference has arisen, can only be determined in pedigree cultures of organisms that are very well understood through decades of genetic analysis. The fact that, whilst there is now a vast body of literature on idiovariations, very little is yet known with certainty, is precisely due to the fact that only very few organisms have so far been studied with sufficient precision.

If we examine the factual evidence currently available on idiovariations with a strictly critical eye, the following picture emerges: in every species of organism that has been sufficiently observed for this purpose to date, individual idiovariations occur, usually for entirely unknown reasons and with highly variable frequency. The vast majority of these are due to the emergence of a new Mendelian fundamental difference. In most cases, when the new idiovariant is crossed with the parent strain, the trait expression of the parent strain exhibits more or less pronounced dominance over the

newly emerging trait. However, there are also a large number of unquestionably valid idiovariants which, when crossed with the parent strain, are dominant. Much rarer, as yet, are idiovariations whose differences from the parent strain are not
'follow Mendel's laws', but are inherited in some other way. This finding is consistent with the fact that, as we have heard, the vast majority of all breed differences follow Mendel's laws, and that inheritance of breed differences according to other laws is only rarely observed.

It is not yet possible to provide specific data on the frequency of idiovariations. Unless a very specific experimental setup is employed, only a small proportion of the cases of idiovariations that actually occur can ever be detected and identified. One can only say that, among the organisms that have so far been studied to any reasonable extent in this regard — namely, *Drosophila* and *Antirrhium* — at least one individual in every 1,000 offspring of a parental pair exhibits an idiovariation. Idiovariations are therefore more common than had long been believed.

When and where idiovariation occurs within an organism, and where the underlying change in the idioplasm takes place, can hardly ever be determined with certainty in higher organisms that reproduce exclusively sexually. However, there is much to suggest that idiovariation can occur at any stage of development and that, accordingly, in higher organisms the vast majority of all idiovariations take place in the diploid cells. Of the greatest theoretical interest here is that, insofar as Mendelian idiovariations are concerned, the two sets of chromosomes in a diploid cell are almost certainly entirely independent of one another. An idiovariation therefore affects only one set of chromosomes.

Idiovariations constantly give rise partly to genuinely new racial differences, and partly to racial differences that already exist elsewhere.

Ultimately, idiovariations form the basis of all phylogenetic development; without them, the emergence of new lineages, races and, subsequently, species would not be possible.

We know almost nothing about the causes of idiovariations. The vast majority of all cases observed in controlled experiments have occurred 'spontaneously'—though this naturally only means that we know nothing about the cause. It can, however, be said that it is probably possible to induce idiovariations through a wide variety of interventions—high temperatures, the effects of chemicals, radium and X-rays, etc.

One factor which, according to all observations available to date, very likely increases the frequency of idiovariations in sexually reproducing organisms is severe inbreeding. A further triggering cause of idiovariations may also be found in cross-breeding between very distinct strains, whereby the entire mechanism of nuclear and cellular division—and above all, reduction division—is disrupted, and the two distinct nuclear components do not fit together properly. However, particularly in the case of idiovariations of this latter category, it is usually very difficult to determine whether an idiovariation or an irregular division is present. Strictly speaking, it is also theoretically almost impossible to make a clear distinction here between idiovariations and mixovariations. The two categories of variations merge into one another here. Idiovariations of this latter type—that is to say, intermediate forms of idiovariations and mixovariations, so to speak—constitute the much-discussed de Vries mutations of Oenothera—at least in the vast majority of cases.

The tendency to frequently give rise to certain idiovariations is very likely inherited as a family trait; it is very likely that this predisposition is inherited in accordance with the laws of segregation. One might imagine this in such a way that certain chromosomes have a particularly unstable structure and therefore give rise to idiovariations particularly easily.

Of great interest to human pathology are experiments conducted to determine whether, and to what extent, severe poisoning alters the hereditary constitution of offspring, i.e. triggers idiovariations. It is very difficult to draw definite conclusions from findings in humans themselves. For if, for example, we find that the children of a chronic alcoholic are in some way deficient, this m a y be due to the fact that the father himself became an alcoholic only because he was already

psychologically deficient; in that case, he would merely have passed on the predisposition he had already inherited from his own ancestors. Alternatively, the children of an alcoholic may be deficient because they grow up in hygienically and socially far more unfavourable conditions than the children of healthy parents. Here, animal experiments show that chronic alcohol poisoning in healthy animals does indeed cause severe damage to their offspring, which manifests itself in a very high number of stillbirths and in weak and, in some cases, malformed young. This does not appear to be merely an 'after-effect' (cf. p. 9), but rather a permanent damage to the idioplasm, for even the grandchild generation (the F_2animals) of the alcohol-exposed animals (the P_1animals) exhibits all these defects, even though the F_1animals were not treated with alcohol. It must therefore be assumed that severe intoxication—and presumably other forms of damage as well—are capable of influencing the idioplasm and causing hereditary damage. It should, of course, be borne in mind that these animal experiments involved levels of alcohol poisoning that are rarely, if ever, encountered even among the heaviest drinkers.

The vast majority of all spontaneously occurring and artificially induced idiovariations, if one were to make a value judgement, constitute malformations and would therefore be unable to survive in the struggle for existence.

In humans, no unambiguous cases of idiovariations are known; however, it can safely be assumed that idiovariations occur here just as frequently as in all other organisms.

In organisms which, like humans, reproduce sexually and through free mating ('panmictically'), the three categories of variation described above constantly occur alongside and intermingle with one another. Determining whether a difference between two people is due to paravariation, mixovariation or idiovariation is usually very difficult—much more so than the layman might initially suppose. What is at stake can—if at all—usually only be determined

a very thorough study. It seems necessary to emphasise this once again, quite particularly, at the end of this chapter.

3. The influence of variations on the composition of a population, the effect of selection processes.

What we today refer to as a population is, from a biological point of view, an immensely diverse mixture. If we were to allow a pair of jackal families from Asia Minor (Canis aureus), a pair of jackal families from North Africa (Canis lupus ter) and a pair of wolf families (Canis lupus) to breed somewhere through free interbreeding under the most favourable conditions , so that a l l m i x o v a r i a n t s are preserved and many i d i o v a r i a n t s—such as dachshund-like legs, pug-like build, greyhound-like build, long-haired coats, etc.—are not weeded out by natural selection, then a canine population would emerge whose diverse composition would roughly correspond to the racial hodgepodge of a modern human population.[4]

It cannot be emphasised strongly enough that what a people—such as the Germans, the English or the French, etc.—have in common and what unites them as a people is not actually 'race', but first and foremost their c o m m o n l a n g u a g e a n d c u l t u r e. Racial differences, such as those between the peoples mentioned, are always merely relative differences insofar as the proportions of the racial components within the various peoples differ somewhat, in one people these racial components are more numerous, in another those are more numerous. Nor do anthropological ethnic boundaries in this sense coincide at all with linguistic boundaries.

If we find that, within a large population numbering many millions, it is virtually never the case that even two individuals are identical, but rather that there is great 'variability' in all characteristics, this is due in part to the fact that individual members vary to different degrees (p. 7), and partly on the fact that, from time to time, individual new idiovariations occur; but, for the most part, this

difference between individuals on the fact that, in such a mixed population, different combinations of Mendelian and non-Mendelian racial differences arise time and again with each act of reproduction. Since paravariations are not heritable, they do not alter the hereditary composition of a population, however great the influence of diet, lifestyle, etc., on the individual may be.

By contrast, a population's hereditary constitution is significantly altered by idiovariations and, under certain conditions (selection processes!), also by mixovariations.

If certain idiovariations, such as the occurrence of a hereditary predisposition to diabetes, were to occur even only occasionally — say, once in every 10,000 births as a heterozygous idiovariation — and if the diabetics were not weeded out by a rigorous process of natural selection, i.e. if they reproduced at the same rate as the population average, then the percentage of diabetics would continue to rise, sooner or later there would have to come a time when the entire population would consist almost exclusively of diabetics. Even the prevention of natural culling—that is, the excessive hygienic and social protection of mentally or physically inferior idiovariants—can lead to the degeneration of a population, unless measures are taken in some way to ensure that the reproduction of the inferior is prevented, or at least is lower than the national average.

For the most part, however, hereditary variation is based not on idiovariation but on mixovariation, that is, on the ever-changing emergence and disappearance of ever-changing combinations. For the most part, these are probably combinations governed by Mendel's laws.

The question now is: what becomes of such a hybrid population over time, and what is its composition after a long series of generations? Let us discuss this question using a simple example and assume for the moment that we have a number of F_1 hybrids between a black (**AA**) and a white (**aa**) breed of rabbit. A

pair of these hybrids (**Aa**) into a large hutch and allow them to breed under the most favourable conditions possible. This is to continue for a number of generations with completely unrestricted mating; in this way, we wish to breed a large population of rabbits, and the question is: what will the composition of such a rabbit population be? The answer is: there will always be almost exactly ¼ of all animals that are white and ¾ that are black. We had placed one female and one male $_{F(1)}$animal in the large cage. Their offspring, that is, the F_2generation, should consist of ¾ black and ¼ white animals. Let us assume, for example, that this generation consists of a total of eight animals, namely:

one homozygous black male **AA**, hereinafter referred to as Male I,
two heterozygous black males **Aa**, hereinafter referred to as Males II and III, one homozygous white male **aa**, hereinafter referred to as Male IV,
one homozygous black female **AA**, hereinafter referred to as female α, two heterozygous black females **Aa**, hereinafter referred to as females β and γ, and one homozygous white female **aa**, hereinafter referred to as female δ.

There are 16 possible matings between these animals, all of which are equally likely and can therefore be treated as occurring with equal frequency, namely:

Female α × Male I will produce 4/4 *n* AA offspring
" α × " II " " 2/4 *n* AA-, 2/4 *n* Aa
" α × " III " " individuals 2/4 *n* AA-, 2/4
" α × " IV " " *n* Aa individuals 4/4 *n* Aa individuals

" β × " I " " 2/4 *n* AA- and 2/4 *n* Aa-animals
" β × " II " " 1/4 *n* AA-, 2/4 *n* Aa-, 1/4 *n* aa-animals
" β × " III " " 1/4 *n* AA-, 2/4 *n* Aa-, 1/4 *n* aa-animals
" β × " IV " " 2/4 *n* Aa-, 2/4 *n* aa-animals

" γ × " I " " 2/4 *n* AA- and 2/4 *n* Aa-animals
" γ × " II " " 1/4 *n* AA-, 2/4 *n* Aa-, 1/4 *n* aa-animals
" γ × " III " " 1/4 *n* AA-, 2/4 *n* Aa-, 1/4 *n* aa-animals
" γ × " IV " " 2/4 *n* Aa-, 2/4 *n* aa-animals

" δ × " I " " 4/4 *n* **Aa** animals
" δ × " II " " 2/4 *n* Aa-, 2/4 *n* aa-animals
" δ × " III " " 2/4 *n* Aa-, 2/4 *n* aa-animals

" δ × " IV " " $_{4/4}$ *n* **aa** animals

Sat $_{16/4}$ *n* AA-, $_{32/4}$ *n* Aa-, $_{16/4}$ *n* aa-animals
i.e. **4** *n* AA- : **8** *n* Aa- : **4** *n* aa- individuals

The results of all 16 of these individual possible matings, each expected to occur with equal frequency, are also shown in this table. The table also calculates the combined outcome of all 16 possible matings, and the result is that the offspring of such a F_2 generation, resulting from free mating, will consist of 1 part AA-, 2 parts Aa-, and 1 part aa-rabbits, just as it would in the case of 1 part AA- : 2 parts Aa- : 1 part aa-rabbits, i.e. an F_2generation produced in this way will have exactly the same composition as the F_2generation. The same applies to subsequent generations, and holds true not only when animals of a single generation mate amongst themselves, but also when individuals from different generations mate, which will indeed be the case in such an experiment. The same calculation can also be carried out if the F_1hybrids differ in several characteristics and split in a more complex ratio, for example 9 : 3 : 3 : 1 or 27 : 9 : 9 : 9 : 3 : 3 : 3 : 1; even then, the subsequent generations will always exhibit roughly the same composition as the F_2generation. If, therefore, a population reproducing through random mating arises from an F_2 generation following a cross, then this population will always exhibit the composition that the original F_2 — generation already exhibited, provided that no selection is taking place.

The case where an entire population — for example, all the rabbits on an island — is descended from a single, uniform $_{F_2}$generation will, of course, rarely occur in nature, but this law applies much more broadly.

For example, we can modify the experiment just described slightly, and let us take the following animals into the large breeding cage as the founding stock for the new rabbit colony: two homozygous black AA males (labelled I and II), two homozygous black AA females (labelled α and β), one heterozygous black Aa male (labelled III

) and one heterozygous black Aa female (labelled γ). If we allow these six rabbits to mate as often as we like and in any order, we will obtain a next generation consisting of homozygous black, heterozygous black and white rabbits in the ratio 25 : 10 : 1.

The calculation yields the following table:

The female α can mate with the male I and will produce:
4/4 *n* black **AA** individuals.
The female α can mate with the male II and will produce:
4/4 *n* black **AA** individuals.
The female α can mate with the male III and will produce:
2/4 *n* black AA and 2/4 *n* black **Aa** individuals.

The female β can mate with the male I and will produce:
4/4 *n* black **AA** individuals.
Female β can mate with male II and will produce:
4/4 *n* black **AA** individuals.
The female β can mate with the male III and will produce:
2/4 *n* black AA and 2/4 *n* black **Aa** individuals.

Female γ can mate with male I and will produce:
2/4 *n* black AA and 2/4 *n* black **Aa** individuals.
The female γ can mate with the male II and will produce:
2/4 *n* black AA and 2/4 *n* black **Aa** individuals.
The female γ can mate with the male III and will produce:
1/4 *n* black AA, 2/4 *n* black Aa, 1/4 *n* white aa individuals.

The result of a l l possible matings: 25/4 *n* black **AA**, 10/4 *n* black **Aa**, 1/4 *n* white aa individuals.

Since all matings have the same probability, a very large number of such matings will result in a population in which the categories **AA**, **Aa** and **aa** occur in a ratio of 25 : 10 : 1, i.e. in the same ratio that already existed in the F_1generation.

If we allow such a population to continue to reproduce freely and without restriction, then all subsequent generations will also exhibit the **same** numerical ratio between the white and the two varieties of black animals. It is thus easy to calculate, for any given initial generation, what composition a resulting population will have.

The prerequisite here is that there is no 'infusion of foreign blood', and that the various categories are **equally** viable and capable of reproduction, so that no selection process takes place.

But what happens if certain specific combinations are constantly culled, or if reproduce at a below-average rate? Let us again take a simple, schematic example: we introduce to an island two female blue Angora rabbits with the genotype **AA XX BB CC dd gg vv** (see p. 36 for the genotype) and two male short-haired wild-coloured rabbits with the genotype **AA XX BB CC DD GG VV**. The F_1generation will then consist of —[5] **Dd Gg** Vv individuals, all of which are short-haired and wild-coloured, and the F_2generation will consist of the following categories in the frequency ratios given in brackets:

— DGV	wild-coloured	short-haired	27
— DGv	"	Angora	9
— DgV	black	short-haired	9
— Dgv	"	Angora	3
— dGV	blue-wild-coloured	short-haired	9
— dGv	" "	Angora	3
— dgV	blue	short-haired	3
— dgv	"	Angora	1

As mentioned, without selective breeding, the resulting rabbit population will remain permanently in this varied composition. Let us now assume that certain categories, such as all blue-wild-coloured Angora animals, i.e. all animals with the genotypes —**dd GG vv** and —**dd Gg vv**, were culled from the breeding stock. Even if this culling of blue-wild-coloured Angora rabbits were carried out very strictly, so that n o a n i m a l of this type were ever allowed to breed, animals of this type would still be born time and again in our rabbit population, because animals with this genetic makeup and physical constitution are produced in such a breeding programme even by parents that look completely different! However, the number of blue-wild-coloured Angora rabbits in the next generation

already be lower. The table below illustrates the effect of such a culling of blue-wild-coloured Angora rabbits.

Without selection, the F_3 generation would exhibit the following categories		in terms of	expressed as a percentage	the elimination of all blue-wild-coloured Angora rabbits results in the ratio (also expressed as a percentage)	i.e. an increase or decrease in %
wild-coloured	short-haired	27	42.18	44.44	+ 2.26
"	Angora	9	14.06	13.82	– 0.24
black	short-haired	9	14.06	14.67	+ 0.61
"	Angora	3	4.69	4.46	– 0.23
blue-wild-coloured	short-haired	9	14.06	13.82	– 0.24
"	Angora	3	4.69	3.19	– 1.50
blue	short-haired	3	4.69	4.46	– 0.23
"	Angora	1	1.56	1.12	– 0.44

As a glance at the table shows, the calculation — which is somewhat cumbersome and therefore not reproduced in full — of the composition of the F_3 generation following such selection reveals that not only has the frequency of blue-wild-coloured Angora rabbits decreased, but that there have also been considerable shifts in the proportions of the remaining categories relative to one another.

If such selection is carried out over a long series of generations, the culled variety will become increasingly rare and the composition of the rabbit population will also be very significantly altered in other respects.

In detail, this effect varies greatly depending on whether dominant or recessive types are culled, whether only one combination or many combinations are culled simultaneously, and so on. It would take us too far afield to go into all the details here. What interests us is that, although certain combinations do not initially disappear completely as a result of such a selection process, the culled types do become rarer and the composition of the population changes in other respects as well.

As explained elsewhere, this has far-reaching implications for the assessment of selection processes in humans. If, in a civilised society, the best-endowed individuals and families—that is, those embodying very specific combinations of traits—consistently reproduce at a lower rate than the average for the population, this will have a disastrous effect on the population as a whole: highly gifted individuals will, admittedly, always emerge once again from the vast mass of the population, but if, over a long series of generations, these highly gifted individuals reproduce any less vigorously than the national average, then these people will become increasingly rare, and eventually such a nation will no longer produce as many 'leading figures' as are necessary to form the backbone of the state and to maintain the people at the height of their culture; such a people will 'degenerate'.

4. The effect of inbreeding.

If inbreeding is practised in an organism that usually reproduces through cross-fertilisation, this almost always entails disadvantages. This realisation of the harmfulness of inbreeding is ancient; a large part of the marriage legislation of even the most ancient civilised peoples is based on it to a greater or lesser extent.

Fig. 13.
Schematic family tree of a family with a hereditary malformation that is Mendelian recessive (such as albinism) — homozygous healthy individuals are shown as white, heterozygous individuals (who are also outwardly healthy) as white with a black dot, and homozygous affected individuals as black. The single case of consanguineous marriage shown (framed by a dotted line) allows for the birth of homozygous affected children.

The harm caused by inbreeding stems from two entirely different factors. Firstly, all forms of inbreeding and consanguineous mating promote the selection of recessive abnormalities. This is best illustrated by the family tree shown in Figure 13. Albinism is inherited in humans as a simple recessive trait. Heterozygous albino individuals appear completely normal externally. If members of such a family with a hereditary predisposition to albinism repeatedly marry into other families not affected by this condition, a proportion of the offspring will always carry the trait in a heterozygous, i.e. latent, form; however, no albinos will be born. Homozygous albinos will only result here if two such heterozygous individuals marry each other. However, the probability of such a case occurring is very high in the case of consanguineous marriage within such a family; consequently, the occurrence of albinism in this family is favoured by consanguineous marriage. A great many families carry a hereditary predisposition to recessive genetic disorders, usually without the family members being aware of it. One family may carry this defect, another that one. Marrying outside the family prevents the hereditary predisposition to disease from becoming homozygous, whilst marrying within the family favours its occurrence. This is one cause of the harmful effects of inbreeding, but not the only one.

A second type of inbred depression is based on the fact that, for unknown reasons, any degree of inbreeding — the closer the relationship, the more rapidly — causes a weakening of the offspring and a reduction in reproductive capacity. This weakening presumably always proceeds to a minimum level reached sooner or later; that is to say, in the case of inbreeding over several generations, the vitality of the offspring initially declines very sharply, then more slowly in later generations, and finally a sort of steady state is reached where further inbreeding no longer causes harm. There is therefore probably a sort of minimum level of vitality that is reached sooner or later through the closest possible inbreeding. This minimum level varies greatly among different organisms.

As regards humans, nothing reliable is known about this effect of prolonged, extremely close inbreeding. Little is known about this in higher animals either. It is therefore sufficient, at the very least, to point out here that, in addition to the first type of inbreeding effects—which have been better studied in humans—there is also a second such type.

Whether the occasionally reported observation that children born of incestuous relationships (children of brother and sister, of father and daughter, etc.) are frequently mentally and physically inferior is due to the 'effects of inbreeding' is highly doubtful. After all, incest generally tends to occur only amongst people who are themselves already severely inferior.

Section Two.

Racial differences among humans.

By

Prof. **Dr Eugen Fischer.**

When considered across the globe in terms of both space and time, human beings are not only different everywhere, but no two individuals are completely alike. Yet within individual groups, the individuals
more similar to one another than to those of other groups. This raises the question of the significance of these differences between individuals and groups. Does the environment—for example, climate, diet or even the social 'milieu'—play a role here? In other words, can those 'traits' that account for the similarities and differences between individuals and groups also change as a result of changes in external factors? Or are there characteristics that are independent of all these influences—referred to as 'peristatic'—which are genetic, transmitted with the genetic material, and are therefore inalienable and unchangeable? 'Anthropobiology' is the name given to the discipline that deals with all these matters. Anthropobiology must therefore investigate, in the case of humans, which of the individual — and which of the group — or, as is usually said, 'racial differences' among humans — are 'paravariations',
'mixovariations' or 'idiovariations'. (See page 6 ff.).

Systematic 'anthropology' has, until now, almost always limited itself to recording these differences anatomically as such, and defining them descriptively and metrically. Naturally, this morphological aspect of research is indispensable for the purposes of racial hygiene as well. But racial hygiene, in particular, must know which of these externally discernible characteristics are hereditary. A purely morphological approach is therefore insufficient; morphological characteristics must be assessed in terms of their origin and their significance for the individual and the group – in other words, they must be considered from a biological perspective. Racial hygiene requires both morphological and biological findings as its foundation.

1. The varying characteristics of human beings.

(Specialised anthropology and anthropobiology.)

There are, of course, absolutely no organs or parts of organs in humans that do not vary. Blacks and Europeans, for example, are distinguishable from one another not only by skin colour, hair type, the shape of the nose, lips and skull, and other characteristics generally recognised as ‘ ’ differences, but there are also corresponding differences in the brain, the muscles and all manner of parts of the internal organs. Indeed, the whole field of anthropology would have to be covered here, with a systematic discussion of every organ system in the human body; this would amount to a veritable comprehensive textbook on anthropology.[6] However, that is not the intention, nor is it necessary. The aim here is merely to lay the foundations for understanding the conclusions regarding racial hygiene that can be drawn from knowledge of the laws of heredity.

a) The Skeleton.

No part of the human body has been studied as intensively or as frequently from an anthropological perspective as the skull. Numerous group and individual characteristics have been identified in all its individual features, as well as in its overall shape and size, using highly refined methods of measurement and notation; consequently, only the most important points can be touched upon here.

For the sake of clarity, and according to their phylogenetic and systematic significance, the cranial characteristics can be divided into individual groups:

Firstly, there is a large number of craniological characteristics (just as there are in all other organ systems), both qualitative and quantitative, by which apes and humans are clearly and sharply distinguished. These characteristics are therefore specific to the human species; they must thus be fixed in the genetic makeup of ‘humans’. By way of example, one might mention: in anthropoids, the small size of the cranial vault in comparison with the facial skeleton, the formation of bony ridges projecting roof-like over the eye sockets (supraorbital ridges), the protrusion of the canine tooth beyond the rest of the dental arch, and the robust development of the lower jaw with a receding, chinless front — in humans, the opposite of all these is present. There can be no doubt as to the heritability of all such human characteristics, nor as to the existence

specific hereditary factors that determine them, and indeed within the gene pool of the entire human race, there can be no doubt. Occasionally, however, an 'ape-like' 'pithecoid' formation appears in humans. One must assume that the common ancestors of humans and apes possessed similar characteristics and that — through circumstances largely unknown to us — such a characteristic reappears as so-called 'atavism', just as, for example, a dark dorsal stripe does in horses. Just as with apes, a number of craniological characteristics sharply distinguish modern humans from Neanderthals — that is, from the entire Homo primigenius (Neanderthal, Spy, La Chapelle, etc.), then from Homo heidelbergensis (the lower jaw from Mauer) and finally from Pithecanthropus erectus (Java). What was stated above regarding the others also applies to these, including the 'atavistic' characteristics. Individual Neanderthal-like characteristics appear here and there as throwbacks to them or similar ancestral forms—and thus as remnants of the ancient genetic pool—but never the complete and truly distinctive combination of Neanderthal characteristics. For the purposes of eugenics, all these characteristics are of lesser importance than the following:

A large number of craniological characteristics may be regarded as racial characteristics in the strict sense. These are, above all, quantitative differences across the entire skull or in individual structures, and also qualitative differences, which occur in numerous combinations and thus help to distinguish between races. The individual characteristic as such may, of course, appear in various combinations with others in quite different races. However, for each characteristic, it must first be investigated whether it is truly heritable, i.e. whether it is a
'racial characteristic'; a great many so-called features are found with particular frequency in certain human groups simply because the same factors determining them affect the entire group. Such features are therefore not racial characteristics.

Racial differences

The most striking racial difference is in the overall shape of the skull, both the cranial vault and the facial skeleton.

In addition to description, measurement is used to determine and record these differences in shape. Since Anders Retzius (1864), one measurement has always been expressed as a percentage of the other, in order to avoid the difficulty of comparing absolute values of two related dimensions — e.g. length and width. This ratio is referred to as an index. For example, the width of the skull is expressed as a percentage of its length (from the forehead to the occiput), and this is termed the length-to-width index. It must be emphasised that the resulting classifications into long-skulled and broad-skulled types, long-faced and broad-faced types, etc., are arbitrary; by no means does the long-skulled nature determined by measurement in one group here and another there automatically imply that they belong together. The relative length, for example, in the long skulls from Sweden is determined by entirely different details of bone formation than in the long skulls from the African countries inhabited by Black people. It depends on the individual shapes of the forehead, crown, occiput, etc. The terms
'long skull' etc. therefore merely provide a brief description of the ratio between two main dimensions.

On the whole, the skulls of each race are characterised by a large number of distinct features, each peculiar to that race. For example, the narrow,

long, low Negroid skull, with distinctly pronounced frontal and parietal protuberances, flat, small nasal bones, distinct so-called pre-nasal fossae, a low, rounded nasal cavity, a roundish, domed, low forehead, a snout-like protrusion of the jawbone (so-called 'prognathism'), and coarse cheekbones, etc., is easily distinguished, for instance, from the large, strongly domed, long, narrow 'Nordic' skull with its protruding, long, narrow nasal bones set against one another like the ridge of a roof, the vertical facial profile, the long, narrow face, and sharply defined chin, etc. This clearly suggests, however, that in all these respects we are dealing with characteristics that are contained within the genetic makeup of the race in question, and which are therefore firmly inherited. And yet certain aspects of their external appearance can be altered by 'peristatic' factors.

Firstly, the 'racial', i.e. hereditary, characteristic shape of the skull, e.g. e.g. its length-to-width ratio—that is, whether a long or a round skull develops—is already distinct in the foetus in the 8th and 9th months, even if the racial differences are nowhere near as pronounced as they are later on.

As is well known, the compressive forces at birth have a deforming effect on the infant's head. This leads to the edges of the bones being pushed over one another and to the bones becoming bent: birth deformity. If the birth does not proceed normally, the deformities become particularly severe. However, these effects are not permanent; they tend to disappear without a trace after days, weeks or months. Abnormally severe deformities, possibly involving fractures or tears in the bones, may indeed result in a permanent deformity, but such isolated pathological cases can be disregarded here.

The fact that the shape of the head, which continues to grow after birth, takes on that of the parents rather than the shape determined by hereditary racial characteristics is, according to general consensus, governed by the inherited growth pattern; the individual parts must grow with a very specific intensity, sequence, speed, etc. However, this growth is directly regulated by the hormones (glandular secretions) of the endocrine glands. The pituitary gland plays a major role here, followed by the gonads. In castrated boys, the cartilaginous suture between the sphenoid and occipital bones remains abnormally open for a long time. Thus, a change in the function of these glands affects skull growth and, consequently, the shape of the skull

. We do not know whether such changes in glandular function are possible during the course of a normal life due to external influences; indirectly, this would also mean that the shape of the skull is influenced from the outside.

> Both phenomena occur under pathological conditions. In cases of pituitary gland disease, we observe (alongside abnormal length growth of the fingers and toes—known as acromegaly, i.e. enlargement of the distal ends of the extremities, possibly to twice their normal size) abnormally vigorous growth of the lower jaw. This is accompanied by the formation of a prominent chin, which protrudes beyond the level of the upper jaw. This condition is known as prognathism (see p. 185 below). Thus, there are clearly peristatic influences at work here, and in some cases it is difficult to determine to what extent the characteristic form is due to these influences and to what extent it is hereditary. Whilst this applies to the lower jaw in the example cited, corresponding effects occur under different circumstances on the skull as a whole. It is well known that the pathological bone growth observed in rickets can lead to entirely pathological skull shapes — caput quadratum.

The developing head, too, can be shaped to some extent by external influences. We shall not consider here the so-called artificial deformation, as practised to a very great extent by Native Americans and other peoples, who constricted the infant's head with small boards or tight bandages. Tightly fastened bonnet straps (Heligoland bonnets, etc.) can also have an effect in the ‘ ’ direction. However, even the way an infant is positioned on a hard or soft surface can influence the growth of the skull in a particular direction and thus produce slight differences in shape. The heads of monozygotic twins, which are otherwise remarkably similar (see p. 86), can be ‘deformed’ in this way to the extent of a difference of almost 8 index units in the length-to-width ratio.

Far more interesting than these interventions – which are, after all, rather crude and whose effects are in themselves easy to understand – are observations of changes in skull shape amongst populations who have left their homeland and migrated to an entirely new environment.

Among the children of Eastern European Jews who emigrated to America, the skull has become narrower than that of their broad-skulled parents; indeed, the later the birth occurred after emigration, the more pronounced this change was. Conversely, the children of Sicilians with narrower skulls in America developed slightly broader heads. There appears, therefore, to have been a shift towards, shall we say, an ‘American’ average. These observations and findings still leave a great many questions to be resolved; as there is as yet no confirmation from other sources, they must for the time being be treated with caution

. But even assuming their full and general validity, they merely show that a certain aspect of skull shape — just as with body height, see p. 103 — is determined by the environment; the rest is reserved for hereditary influence and constitutes inalienable genetic material. It is not yet entirely possible to draw a clear distinction between these two factors; perhaps these relationships also explain some of the unclear findings in the study of races in Central Europe.

Finally, one last influence of external factors on skull shape must be mentioned, albeit an indirect one: that of body length. As body height increases, the longitudinal growth of the skull increases slightly more or more rapidly than its width; thus, as body height increases , skulls become somewhat more elongated, resulting in a slightly lower length-to-width ratio. One can always expect a corresponding change in the index values as body height increases. Now, as will be shown below (p. 104) (apart from certain pathological cases), the height of an individual is partly determined by their nutritional and perhaps other conditions during the growth period. Consequently, the resulting increase or decrease in height will also be accompanied by a corresponding decrease or increase in the index. When assessing an individual's length-to-width index, one must therefore take their height into account.

Just as these circumstances influence the cranial structure, so too can social circumstances exert some influence on the facial skeleton. The socially higher and lower strata of the population within the same race differ: the former have finer, narrower faces, the latter coarser, broader ones; this is the case here just as it is in Japan. This applies not only to the soft tissues but also to the bony structure. The differences are, however, very slight, and one must be very careful not to mistake genuine racial differences for something else when one thinks one sees them, for often, even within one and the same population, the upper and lower social classes are not of the same racial composition.

Finally, attention must be drawn here to a group of cranial characteristics which, taken as a whole, are determined by hormonal action—specifically from the gonads—namely the sexual characteristics of the skull. Experiments on animals have amply

demonstrated that these characteristics depend entirely on the 'internal secretion' of the gonads; what we know about the consequences of castration in humans does not contradict this. For theoretical reasons, we may readily apply these findings to humans.

Thus, the distinctive features of the male and female skulls are determined in this way. The male skull is larger, heavier, and the muscular ridges are more pronounced. The lower jaw is more robust, and the teeth are larger. In the female skull, the frontal and parietal protuberances are usually more pronounced than in men, whilst the glabella and supraorbital ridges are usually less developed, and so on. The differences are consistent across all races; like all secondary sexual characteristics, their degree of development varies greatly from individual to individual.

For the craniological characteristics discussed so far, the influence of non-hereditary 'peristatic' factors has consistently been observed. At times this parakinesis has, so to speak, reigned supreme; at other times it has merely modified hereditary development to a greater or lesser extent, and here and there perhaps only minimally. Yet one must not allow oneself to be led astray by these numerous cases of established environmental influence into underestimating, or even overlooking, the great power of the genetic endowment. Through careful observation and reflection, we are nevertheless led to assign the primary role to it. All these modifying influences act only upon a form which possesses within itself the capacity to develop in a very specific direction, towards a very specific size, shape or number. The paratypical deviations are merely superimposed, so to speak, upon this ideally aspired-to development. We observe in large groups of people, where one can study thousands of individuals living simultaneously under quite different conditions, that the shape of the skull is the same, that a very specific racial form persists. Not, of course, in such a way that all individuals have exactly the same shape down to the last detail, but it does fluctuate regularly around a typical mean! We therefore have typical index values for the individual races. The peculiar distribution of the length-to-width index across the globe, or specifically in Europe, is in itself proof that it is a racial characteristic and a manifestation of race. Only in this way can this distribution be explained in detail (see p. 124, Anthropography of Europe). This heritability—that is, the

tenacious persistence of the racial form of the head, is particularly evident where we can trace the fortunes of peoples, mass migrations, the formation of states and the collapse of states over long periods of history or prehistory. In Egypt, this 'constancy' of forms could be observed; in the Near East, it was demonstrated that in 'refuge areas'—such as inhospitable mountains, deserts, marshlands, etc.—the ancient types remain genetically unchanged over millennia, whilst all around them new immigrants are weeded out, so that the ancient race
'restores itself'. It must therefore be emphasised with the utmost vigour: it is precisely the observation that certain variations in skull length, width or height are peristatic in nature—that is, modifiable—has only served to reinforce the fact that the form in its very foundation, so to speak, the form itself, is hereditary, fixed and inalienable.

> It is almost ridiculous to have to point out that if, for example, a person's skull—due to a different diet for the child, a different sleeping position for the infant, or other factors—actually has a length-to-width ratio of 76 instead of 74, they would not belong to a different race simply because we call them mesocephalic, whereas they would otherwise have been dolichocephalic! Yet there are still people for whom these terms are fixed labels, through which only then is a skull assigned to a very specific race!

What has been said here regarding skull shape applies equally to facial shape, to the shape of the eyes, the nasal cavity, the palate, etc. (For details on these matters, insofar as they specifically concern the face, see p. 107). These, too, are largely determined by heredity; here, too, the study of races—particularly in the case of relatively isolated races or tribes such as the Eskimos, the Wedda, the Hottentots, etc.—provides us with such typical examples of the racial distribution of certain forms that one cannot doubt their hereditary nature.

Under these circumstances, it is hardly surprising that the anthropological measurements of the heads of identical twins show an extraordinary similarity. In adult identical twins, a 23-year-old girl (Jewish), the following measurements were found:

	1. Maximum head length[7]	3. Maximum head width	4. Minimum frontal width	6. Zygomatic arch width	8. Lower jaw angle width	18. Morphological facial height	21. Height of the nose	13. Nasal width
I.	191	150	107	130	89	106	48	30
II.	190	149	107	130	91	106	47	30

The head length-width index for the two is 78.5 and 78.9 respectively; the absolute differences in head length and width are only 1 mm each (repeatedly and precisely measured!), whilst forehead width, maximum facial width and facial height are identical to within fractions of a millimetre! Only heredity can achieve this; even the same environment could not affect two individuals so perfectly alike. The width of the mandibular angle differs the most, namely by 2 mm, and one can easily imagine that external factors would be most influential here. But on the whole, one must — I would say with utter amazement — note how little external factors appear to have shaped the skull here!

The most conclusive proof of the hereditary transmission of these characteristics would be the observation that Mendelian segregation occurs in them. This does indeed appear to be the case. It is extremely difficult to provide absolutely precise proof. It is already clear from what has been said that external influences may have a somewhat modifying effect on all these characteristics; thus, minor deviations in the indices are to be expected, which distort the pure picture of Mendelian figures. Nevertheless, probabilistic evidence for their validity has been established. Firstly, in typical, verifiable mixtures of two different races, it can be seen that the mixed population does not possess a cranial form whose length-to-width index fluctuates around a single mean value; rather, the variation curve remains bimodal, and the two original mean values can still be discerned, as is the case with Native American mixtures in the Great Lakes region of North America and with the 'bastards' of German South-West Africa. In the case of the latter, the relationships have been examined in great detail; it transpired that they correspond exactly to those we observed in Mendelian inheritance according to the N i l s s o n - E h l e type (see above, p. 43). The index values of the children exceed those of the parents, and the total values of all the children exceed the limits of all the parents. This also applies to European data. Consequently, it seems highly probable that we must assume there are several (not just one) hereditary factors for head length and several for head width (not, however, for 'shape'). The larger dimension is always dominant over the smaller one. All this also explains certain findings regarding skull shapes, such as those found in the German population, where all the intermingling resulting from the Migration Period and continuing to the present day has not produced a single average skull shape, but rather has consistently 're-manifested' the old forms. As regards facial width, too, one can

it is highly probable that Mendel's laws apply. Mixed-race individuals of varying degrees between narrow-faced Caucasians and broad-faced Native Americans do not exhibit an intermediate facial width, but rather sometimes one parental form and sometimes the other; the same applies to mixed-race individuals between Hottentots and Boers. There is no question of a fusion, of a form determined solely by the environment; both parental forms continually reappear side by side. The width of the forehead, relative to the zygomatic arch width (fronto-jugal index), also clearly shows a Mendelian divergence of values in the children compared with their parents (African mixed-race individuals). Although it has not yet been possible to provide numerical evidence of Mendel's law for these index values in the same way as has been done for colours, flower shapes, hair types, etc. , the overall behaviour of the indices in mixed-race populations—the complete absence of balanced mean values, the transgression of mixed-race values beyond the parental limits, and the constant recurrence of parental values—clearly points to their hereditary nature. We may therefore record the following as an established finding: the primary characteristic of skull and facial shape is hereditary and unchangeable, whilst only their dimensions exhibit a slight deviation, either above or below, from this primary characteristic; this deviation is 'parakinetically' determined, that is, caused by external influences (peristase).

b) S k e l e t o n (excluding the skull).

A great many differences can be found in all individual bones, varying by race and individual, which cannot be discussed in detail here. Most have at least a very strong parakinetically determined component. The sturdiness of the bones of civilised humans compared with those of so-called 'savages' is likely to be due chiefly to dietary conditions; the curvature of the femur and tibia, and the thickening of these two bones from front to back, are likely to be due to the functional strain involved in squatting, rather than being hereditary. The same probably applies to all manner of other characteristics; even if we cannot demonstrate the causal relationships, for example, the enlargement of the anterior aspect of the lumbar vertebral bodies in Europeans compared with Africans or Australians. By contrast, the differences in the shape and size of the pelvis observed among the various races are likely to be hereditary.

c) M u s c u l a r s y s t e m .

Although differences in the frequency of the numerous variations found in the muscular system have been demonstrated across different races, it is highly doubtful whether such variations are hereditary.

d) Internal organs.

Exactly the same applies to most of the differences observed in the frequency of certain structures in the digestive, respiratory, circulatory, urinary and reproductive systems. These include variations in the behaviour of wisdom teeth, diverse cusp formations on the teeth, the number and arrangement of certain tongue papillae, lung volume, intestinal length and many other features. The absence or atrophy of the upper lateral incisor, for example, is frequently observed within families, so that one must assume a hereditary component; for the other structures, no definite conclusions can be drawn.

e) Neurological system and sensory organs.

The brain has always aroused particular anthropological interest due to the great diversity of its mental capacities, as measured by cultural development. As an anatomical expression of these differences, a marked variation in brain weight among individual human groups can be observed. Even when body size (which is not without influence) is taken into account, the average brain weight among Europeans, Chinese and Japanese is found to be considerably greater than among Africans and Australians. Within a race, on average (despite many individual exceptions), individuals with high intellectual abilities have heavier brains than the average for their people. — The convolutions and sulci of the cerebral cortex vary so much from one individual to another that no racial differences can be detected. (The same applies to apes such as chimpanzees.) Whether individual forms of convolution are inherited is doubtful.

There can be no doubt that intellectual aptitudes and abilities are clearly inherited — although life then shapes these aptitudes, leading to their development or atrophy . The fact that we cannot observe hereditary differences in the brain corresponding to such inheritance is, given the current state of our knowledge, self-evident.

As regards the sense organs, the following should be noted: the olfactory organ as such is, as is well known, greatly reduced in humans—and indeed already in apes; by contrast, the 'external' nose is a typically human structure; it varies greatly between races and should probably be regarded as one of the best racial characteristics, because its numerous variations, as far as we can judge, are purely hereditary. (For further details, see the chapter on Physiognomy, p. 107). In the eye, racial differences are also found in the nervous system. East Asians have a flatter fovea and a larger rod-free zone; the finest and most densely packed cones are found in Hottentots, followed by some Indians, then Malays; the Melanesians have the least refined cones, followed by Europeans. These differences must surely be hereditary.

The most conspicuous variation in the eye, namely the different colours of the iris (see p. 98), has been the subject of the most research.

Of particular interest in the eyelid apparatus is, amongst other things, the 'almond-shaped' palpebral fissure, i.e. a fissure with a rounder medial and pointed lateral angle — in contrast to the more common, general spindle shape with two equal angles. It is hereditary; it occurs most frequently in the Near East and reappears generation after generation, for example in the Jewish population. In most cases, this almond shape is associated with an oblique position of the palpebral fissure, such that the outer corner of the eye is higher than the inner one. This is, for
found frequently among the Hottentots, for example. And for this trait, Mendelian segregation has been demonstrated (in mixed-race individuals between Hottentots and Europeans). The 'oblique' trait is recessive in inheritance relative to the 'straight' trait. The inner corner of the eye is of further interest.

> A malformation occurs here in the form of a membranous connection between the upper and lower eyelids, such that the inner corner of the eye is bridged by an arched fold, known as the epicanthus. As mentioned, this is a malformation of no anthropological interest. However, it resembles (and has been confused with) a typical racial characteristic, the so-called ' ' or 'Mongolian fold', a variant typical of the Chinese, Japanese, Kalmyks (in short, the Mongoloids), and also the Eskimos and Malays.

The so-called Mongolian fold is a loose fold of skin on the upper eyelid, which is longest towards the nose, becoming shorter towards the outside, and gradually tapering off above the centre of the eye or even further out, extending down over the upper eyelid margin. As a result, the inner corner of the eye is covered, as is the corner itself, including the lacrimal papilla; towards

the outer edge of the eyelid gradually becomes exposed; the outer corner is always exposed. This apparently creates a new inner corner of the eye (between the edge of the fold and the edge of the lower eyelid), which sits slightly lower than the 'normal' one, causing the transverse axis of the palpebral fissure to slope upwards — the 'slanted' eyes of the Mongols — the palpebral fissure also becomes particularly narrow towards the nose,
'slit eyes'. If one pulls the skin of the upper eyelid upwards with a finger, an eye and eyelid appear that are entirely similar to those of a European. — The eye of the Hottentot possesses a completely analogous structure, without one being permitted to assume a hereditary connection. In interracial crossbreeding, the formation of folds appears to be dominantly inherited, but systematic studies are lacking.

With regard to the ear, of interest are cases where development is arrested at stages it would otherwise undergo, such as the absence of the rolled-back rear edge, the presence of a pointed tip in place of Darwin's tubercle, etc. A quite peculiar form with pronounced multiple indentations along the edge of the ear occurs (only) among the Bushmen and is clearly inherited.

f) P i g m e n t a t i o n .

Skin colour is primarily determined by pigment granules in the epidermis. The less pigment there is, the more the blood colour of the richly perfused subcutaneous tissue shines through ('incarnate' of the
'white' European).[8] The pigment granules are the same in all races, located in the deepest layers of the epidermal cells; however, their quantity and colour vary greatly between races.

> The first pigment appears during the embryonic stage. At birth, pigmentation is far from complete. The Black infant is not black-brown, like its parents, but has very little colour to begin with, so that the skin is slate-grey or a dirty whitish-brown. In newborns, the skin is dark red due to the rich blood supply. It is only during the first few months of life that the pigmentation characteristic of the respective race develops.

Apart from this pigment, the human embryo has very scattered pigment cells in the dermis, which are slightly more numerous on the back than on the front. In all Mongoloid peoples—including Eskimos, Native Americans and some South Sea tribes—these pigment cells are clustered at the lower back, above the gluteal cleft, to such an extent that a pigmented spot the size of a penny to

taler-sized, or even palm-sized, patch. As the brown pigment lies deep beneath the skin, it shimmers through with a bluish tinge. ('Blue spot' or Mongolian spot). The patch disappears during the first few years of life. It is, of course, reasonable to assume that the Mongolian spot is determined by specific genetic factors.

> The pigment granules of the various races appear to differ. Sometimes, it is likely that only a greater quantity of the same pigment causes a stronger colour intensity. But in other cases, there is certainly also a qualitative difference. Mongoloids have yellow or yellow-red pigment. Americans (Native Americans, Eskimos) are similar or identical to them in this respect. However, dark pigments are apparently often present as well. Perhaps Black people also have two pigments. The fact that, following interbreeding with Africans and subsequent sustained reproduction with Caucasians, even after several generations the pale crescent-shaped marks at the base of the thumbnail and, here and there, the nasolabial fold on the cheek are still yellow in colour, whilst there is no longer any trace of brown colouring anywhere, suggests a difference between the brown and yellow pigments. The racial difference in pigments, and the apparently quite distinct chemical mechanisms of their formation, are likely to account for the great complexity of colouration in racial hybrids.

This raises the question of how non-hereditary factors influence this picture. The main factor to be mentioned here is the effect of sunlight, particularly that of ultraviolet rays. Their influence stimulates the epidermal cells to produce excessive pigment. However, the ability to react in this way varies greatly between breeds in hereditary terms. In almost all races, this stimulus results in rapid, uniform pigmentation, in the manner characteristic of the race in question, which in turn protects the underlying tissue—the network of blood vessels—from the damaging effects of the rays. This protection is, of course, relative; even the tanned skin of a Southern European offers less protection against the tropical sun than, for example, that of an African! In the
'Nordic' (see below) fair-skinned, blond race, on the other hand, this reaction is almost entirely absent and, where present, proceeds very slowly. Consequently, the unchecked rays damage the skin; it becomes inflamed, resulting in sunburn (erythema solare), and the damaged skin peels off in flakes. Only very prolonged and frequent exposure to the sun produces even a very slight tan on such skin. —

This 'acquired' pigment is then lost again — in all races
— when the factor causing it, namely light, is removed. The sunburnt European pales again in winter, but even the Black person deprived of the tropical sun becomes noticeably paler than they were in their homeland.

All other influences are of lesser significance. Worth mentioning are prolonged exposure to heat (blast furnaces, baths, mud packs), the temporary facial discolouration in pregnant women, and discolouration caused by adrenal gland disorders, amongst others.

What we therefore see as 'colour' in humans is, firstly, inherited pigmentation and, secondly, the random state of the reaction to exposure to light or bleaching. Paratypical colouring thus modifies the idiotypical. It is therefore incorrect to compare, for example, the skin of a European—artificially bleached by wearing clothes—with that of a naked 'person of colour'.

The inheritance of skin colour is a established fact, even if not all the details can yet be interpreted. A clear splitting of traits clearly takes place. In cross-breeds between light and dark skin, there are never intermediate shades that remain constant. Whites and Blacks produce medium-dark mulattos. However, their offspring split into different types amongst themselves. Based on new and precise studies carried out in Jamaica and Bermuda, it may be surmised that at least two genetic factors cause the brown component in Black skin and a further one the yellow component. Their combinations largely explain the marked variations in colour among mixed-race individuals of varying degrees and in backcrosses. In addition, there are likely to be genetic factors governing skin thickness, etc. The frequently heard claim that all mulattos are of an intermediate brown colour does not stand up to the precise studies based on around 100 family trees from those islands. Heredity is therefore a division based on a series of factors.[9] The same occurs in the interbreeding of Europeans with Hottentot women. Here, too, there is a divergence into numerous colours. On the whole, light-coloured individuals predominate conspicuously, suggesting that some light-colour factor is likely to be dominant. The peculiar phenomenon whereby Tamil-Malay mixed-race individuals are often darker than both parents, and whereby mixed-race individuals of Portuguese and Indian descent, when bred amongst themselves, can develop an almost jet-black colour in subsequent generations, can be explained by corresponding results from cross-breeding experiments in animal breeding (polymery).

The interbreeding of fair-skinned and dark-skinned individuals in Europe requires more detailed investigation in certain respects; as far as the available data indicate, there is also a clear divergence into individual colours here, and a whole number of hereditary factors must be assumed in this case as well.

A lack of colour resulting from the absence of certain factors is known as albinism (see p. 97).

g) Hair colour.

The variety of human hair colours is due not only to pigments but also to structural factors. The pigments present include pigment granules and dissolved pigment. The granules are found in the cortical cells, and also between them. In some cases, they are present in only a few cells (light hair colours); in others, they occur in dense quantities in all cells (dark hair colours). The medullary cells may also contain pigment granules. Diffuse, dissolved pigment has been definitively detected in red pubic hair and in the yellowish-blonde hair of albinos; it may be present in all red hair.

The air content and oiliness (sebaceous glands) of the hair are significant factors in determining hair colour. Furthermore, sunlight and seawater have a bleaching effect, so one must always bear these paratypical influences in mind.

As is well known, pigment loss occurs with age; the hair turns grey, then white, due to the formation of gas bubbles and the loss of pigment. It is known that this phenomenon begins at varying ages; indeed, it even appears that early or late greying is hereditary in certain families; it would be necessary to establish this precisely at some point.

All these factors, in their manifold combinations, produce an extraordinarily rich spectrum of colours across the whole of humanity. From the lightest yellowish-white and the most delicate silver-grey, the spectrum extends in two parallel series down to black: one via golden blonde, golden brown and brown to the deepest black-brown; the other via silver-grey, ash-grey and dark grey (not age-related greying) to true black. The first series always has a reddish tinge to its colour.

It has been shown that the vast majority of all human races have dark black-brown and black hair. Lighter brown and blonde were originally found as racial colours only in Europe.

Inheritance follows Mendelian segregation. Even without specific figures, the following evidence would be conclusive. For well over 1,000 years, people with blonde and brown hair have been intermarrying in Europe, and the result has not been a uniform medium brown or light brown; rather, even today, bright light blonde still appears alongside dark brown. And for countless generations we have seen black-haired and red-haired Jews intermarry; the result has not been a uniform dark reddish-brown. But statistics on individual cases have also shown this: the hair colours of the children of a marriage are not all the same—a mean of the two parental colours—but vary greatly. This clear alternation in inheritance, this complete absence of intermediate inheritance, is particularly compelling evidence of the constancy of racial characteristics, of their preservation across endless generations, and of the possibility of breeding for or eliminating such racial characteristics.

But this alternation also holds true in individual cases, as observation of extensive statistical data demonstrates. Children are generally no darker-haired than the darker of their parents. Yet this does occasionally occur; it is explained by a combination of factors (see p. 35). (Similarly, the interbreeding of a fair-haired European with a light-haired Negro albino produces a typical dark-skinned mulatto.) Conversely, the children's hair colours are often lighter than that of the lighter-haired parent. The darker colours mask the lighter ones; light hair is inherited as a recessive trait in relation to dark hair.

What has been observed here in European children has also proved to be true for mixed-race offspring of Europeans and Africans, and of Europeans and Hottentots.

One must therefore assume several factors for the inheritance of granular pigment; in line with our observations in animals (rabbits, mice, etc.), a basic factor for pigment formation, A; individuals with aa have albino-like white hair (see below). Then there are several (Z, Y, X...) factors for the formation of brown pigment, each of which intensifies the colour produced by the preceding factors. Parallel to these — perhaps corresponding to the 'grey' series, such as K, J, H... — are factors that determine the equivalent for this series. In the first series, the red factor(s) is (are) also added. We may regard this, alongside the brown factors, as a distinct factor which, without altering the intensity of the colour, can be combined with any of those

factors; it is a red-inducing factor R (perhaps the formation of diffuse pigment?). Red is then dominant over non-red. The individuals of the 'grey' series are all rr individuals. In light to medium colours of the 'brown' series, the absence or presence of red manifests as gradually varying degrees of redness; in contrast, darker granular pigment will mask the red colour.

These genetic relationships are likely to account for the varying frequency of red-haired individuals. Whether the occurrence of isolated small groups with a particularly high prevalence of red hair, for example amongst the Melanesians of New Guinea, should be regarded as a spontaneous manifestation of the red gene or as the loss of factors that previously masked or rendered it inactive remains an open question.

A similar principle applies to the distribution of blond hair across a large race such as the 'Nordic' race. Incidentally, we recognise only this single race as truly blond.

The phenomenon observed in Baden, Denmark, Scotland and amongst the 'Bastards' in South-West Africa—namely that the female population has darker hair than the male population—is probably due to the fact that certain pigment traits are localised in the sex chromosome. Since the human female is homozygous in this respect, more female individuals must inherit these dominant colour traits than males. It also follows that men more often inherit the pigment allele from their mother than from their father. This corresponds to an old popular belief, which, however, should one day be statistically corroborated: dark-haired fathers and blonde mothers are more likely to have blonde sons and dark-haired daughters than the reverse.

The same applies to brown eye colour, at least in certain cases. Among Swedes, Finns and Lapps, the prevalence of brown eyes is so much higher in women than in men that one must necessarily assume, as above, a sex-linked inheritance pattern.

A striking feature of human hair colour is what is known as 'darkening with age'. Even a cursory observation shows that many fair-haired children later develop dark hair. A more detailed investigation reveals that among several groups with dark hair, such as
for example, the Japanese, South American Indians, and perhaps the Papuans (Africans have not been studied), young children are a shade lighter than adults; this would be the same phenomenon for hair as for skin, with pigment increasing slightly during childhood. But this is not the 'darkening' found in Central Europe. Here, many children are light blonde, golden blonde, reddish blonde, occasionally even quite yellow-blonde —

and as adults, the same individuals are brown, dark brown, indeed black-brown. This remarkable phenomenon of a complete change in hair colour is probably best explained by classifying it among the cases of so-called 'dominance reversal'. A marked darkening is therefore a consequence of interbreeding. Among the ancestors of the individuals in question there were certainly both blond and brown-haired people; initially the blond factor dominates, followed by the brown factor (to put it briefly). Among the mixed-race population in German South-West Africa—the ' ', the descendants of fair-haired Boers and black-haired Hottentot women—all the young people are fair-haired (often with the typical frizzy Hottentot hair), whilst the adults are for the most part dark-haired, even black.

Finally, attention must be drawn to the lack of hair colour. In cases of partial albinism, individual tufts of white hair are found. This is often associated with albinotic patches on the skin. Among coloured races, the condition occurs at least as frequently, though it is naturally much more conspicuous there. This form of albinism is inherited dominantly. It must therefore be assumed that there is an inhibitory factor causing the lack of pigment in the individual areas. A number of family trees are known in which this condition has been traced across several generations. In contrast to this is total albinism. The body produces no pigment at all; the hair is white, the skin pale white, and the iris is pigment-free, appearing translucent red. This lack of colour is a defective idiovariant; in other words, the basic factor for colour A is missing. This form of albinism is inherited in a recessive manner. A number of pedigrees are known. The condition also occurs at least as frequently in people of colour as it does in us. Incomplete albinism is also quite common, or it improves somewhat, so to speak, in adulthood. The white hair is, or becomes, a pale yellowish colour; the skin is (in the case of African albinos) a dirty white or yellowish white. One or the other factor is present after all and, because it is alone, is too weak to be expressed until later.

Let us now consider, alongside the occurrence of idio- and mixovariations—that is, phenomena subject to heredity—the effects of peristasis. Do paravariations occur?

Certainly not any distinct or pronounced ones. The slight bleaching of the hair caused by sun and water can be disregarded here; moreover, we do not observe any direct colour-altering influence of climatic factors on the hair, nor any caused by diet or other factors. (Apart from ageing and, possibly, sudden [shock-induced] bleaching.) The distribution of hair colour across the globe does not correspond consistently

climatic zones. In this respect, therefore, hair colour constitutes a distinct racial characteristic.

h) Eye colour.

The iris of mammals owes its colour to a dual pigmentation. The rearmost layer of the iris consists of a double layer of intensely coloured epithelial cells (retinal epithelium), which alone is sufficient to make the iris light-tight against the interior of the eye. In front of this, the iris tissue contains a multitude of large and small branched pigment cells, which often fill the iris entirely. There is only one exception to this structure, which is common to all mammals: in some light-coloured breeds of various domestic animals, the iris loses some or all of its pigment cells in the anterior tissue; as a result, the iris appears light yellow-brown, grey, green or blue. (The latter occurs when the pigment cells have completely disappeared; the pigmented iris epithelium, which always remains, then shimmers through the semi-transparent anterior layers of the iris, causing the blue colouration.) In humans, the colouration is generally the same as in mammals; however, depending on race—just as with those individual domestic animal breeds—the pigment in the anterior layer of the iris may also be lost. Thus, there are human eyes of exactly corresponding shades of light blue, grey, green and blue. The same loss occurs on an individual basis in cases of partial albinism; numerous such cases are known, for example among Black people, who then have blue eyes with fair skin and yellowish-white hair. In complete albinos, the loss of pigment may go one step further, affecting the posterior iris epithelium as well; the iris is then translucent and appears red due to the colour of the blood shining through.

It is reasonable to assume that the anatomical absence of the anterior iris pigment is due to a defective variation. Accordingly, the inheritance of light eye colours is to be regarded as a recessive Mendelian trait. Observation confirms this and shows that the loss of (at least) one genetic factor results in light brown or grey eyes, whilst the loss of a further factor turns these into blue eyes. Parents with p u r e blue eyes invariably have blue-eyed children. The brown eye colour — which is heterozygous — can mask grey, green and blue, and the children of parents with that eye type may therefore exhibit all these colours. Couples with

dark brown eyes will either have only children with the same eye colour (as is the case, for example, with all people of colour; they are homozygous) — or some children will have light-coloured eyes, meaning at least one parent was heterozygous.

The fact that eye colour — (even in people of colour?) — sometimes only fully develops a few months after birth, and that the eyes may become slightly darker later on, is probably linked to the overall process of pigment formation. Also relevant here is the fact that, in old age, the eyes become lighter again; in the case of brown eyes, a slight discolouration probably occurs, whilst in blue eyes the iris tissue becomes denser, causing the coloured layer to show through less, and the dense connective tissue to appear pale and watery-whitish.

The so-called 'white of the eye' is white only in us Europeans; in all non-white races, it is a dirty grey, yellowish in colour with light brown-yellow marbling. The deeper cell layers of the conjunctival epithelium are always pigmented here (as in all mammals, with the exception of very light-coloured domestic breeds).

It would appear that external factors have no influence whatsoever on eye colour, meaning that this too can serve as a reliable breed characteristic.

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Finally, mention must be made of what is known as 'complexion'. This refers to the uniform colouring of the skin, hair and iris. In the vast majority of individuals, including those of mixed race, the relative degree of pigmentation at these three sites is consistent. It would therefore seem that the same genetic factors are responsible for pigmentation at all these sites simultaneously. However, the fact that there are also factors which influence each of the three sites individually in this respect is demonstrated by cases of disharmonious colouring, which are not infrequently observed.

Little attention has yet been drawn to the fact that here in our region, among black-haired (and black-eyed) Jews of the Sephardic type, a particularly pale, low-pigment skin is almost the norm (though is this also the case in Southern Europe and North Africa?).

Similarly, the difference in colour between head hair and body hair (pubic hair and beard hair, which correspond in this respect) suggests that, here too, individual factors affecting pigmentation must be assumed to exist alongside those common to the ' '. In our part of the world, men with brown head hair very often have lighter or reddish body hair, but almost never the other way round. And with blond head hair, the body hair is also blond, or possibly red. In contrast, blond women very often have darker pubic hair.

i) Hair shape.

Whilst almost all wild mammals have smooth, simple hair or, if more woolly, at least a uniform coat within the species in question, humans exhibit, depending on their race, a very

variety in their hair types. It is only in domestic animals—and specifically in the majority of those species—that we find the same conditions (woolly coat, angora-like coat).

> One may well assume a hair type in which the hairs hang down soft and straight; they lie close to the body's surface, thus flowing down from the head in a soft cascade. This type is called 'straight' hair. It is typical of Europeans, Australians, the so-called Wedda people (Wedda in Ceylon, Senoi in Malacca, etc.), the Ainu and others. From this hair type, the thickness and coarseness may increase, and the hair becomes 'stiff', as is the case with Mongoloids, Native Americans and Eskimos. On the other hand, the slight curvature may intensify into multiple wavy bends, resulting in the
> 'wavy' hair type (with wide and narrow waves). Finally, a spiral twist can produce curls; where this spiral twist is tighter, the result is the typical 'frizzy' hair, in which the individual hair spirals may clump together to form a dense, mattress-like pad (common among many Africans and Melanesians). Finally, the spirals may be wound very tightly, with the hair being short and the spirals of neighbouring hairs each braided together into small clumps; this is referred to as 'fil-fil' or peppercorn hair, as found among the Bushmen.

It has been shown that, both in cross-breeding between straight-haired and frizzy-haired Europeans, and in cross-breeding between Europeans and Hottentots and between Europeans and Africans, straight hair in both parents results only in straight-haired children. One may assume that straight hair serves as the base, to which is added the 'curling' factor C, which was previously absent; straight hair is therefore cc. Parents with loosely wavy or curly hair, as well as those with tightly crimped hair, have children with all these hair types. Those with loosely wavy and curly hair are heterozygous (C c). It is probably safe to assume that the actual spiral twist is determined by a second factor; the first factor merely causes a bend (wave), whilst the second (S) transforms this into, or adds to, the spiral twist; the tightest frizzy hair would then be CC SS hair ( ), whereas straight hair would be cc ss.

According to a study of Chinese-Filipino mixed-race individuals, coarse hair appears to be dominant in such a mixture, unlike the straight hair found in Europeans. One might therefore assume the existence of a 'coarsening' factor which is absent in straight hair (and frizzy hair).[10]

Hair growth as such—that is, the distribution and quantity of hair over the entire body—varies considerably between races. The boundaries of scalp hair are generally the same. Heavy body hair, which is not particularly noticeable among us, is found not only among Northern Europeans and Mediterraneans but also among certain groups in the Near East, as well as the Ainu and Australians. All other races, by contrast, have less body hair; the American Indians and Eskimos have the least.

The geographical distribution of these phenomena argues against the existence of external influences; these must be characteristics rooted in the genetic makeup of the races in question. The same applies to the density and shape of the beard. Among the Japanese, the beard appears later than it does among us Europeans; yet among them, as well as among Africans and Melanesians, it takes the same form as it does here. In contrast, the beard of the Wedda covers only the underside of the chin and its tip; it has a goatee-like shape, whilst the cheeks and lips remain entirely or almost entirely bare. Here, too, we are evidently dealing with individual characteristics dependent on specific hereditary factors. The same applies to the individual formation of the fused eyebrows above the nose, which occurs frequently, for example, amongst people of the Near East and on Crete.

k) S k i n.

On the surface of the skin, we find wrinkles and creases in many places, and in some cases also furrows and grooves. The wrinkles and creases are folds and creases in the skin where it is regularly bent or folded by the action of the muscles. The flexion creases on the palm of the hand and at the bends of the fingers—the so-called 'M-shape'—develop even before birth as a result of the embryo's movements. The marked individual variations are due to countless minor differences in the arrangement of the muscles, the position of the joints, the distribution of small fat pads beneath the skin, and so on. Exactly the same applies to the wrinkles on the face. They are not hereditary. In contrast to these wrinkles, the skin ridges and papillary ridges form distinctive fine patterns on the palms and soles, consisting of fine, arched ridges between which equally fine grooves run. On the fingertips, the ridges form intricate patterns, arched designs, spirals or double spirals, etc., from which lines of roughly parallel ridges radiate. On the foot, they are essentially the same as those on the hand, but are greatly reduced in detail.

These tactile patterns correspond to the tactile or ' ' pads on the soles of monkeys and other mammals; in children, they develop as proper pads. Between any two of the metacarpal pads, the lines run in such a way that the boundary lines form a so-called 'triradius'. Two of its rays encircle the base of the fingers, whilst the third runs into the palm and terminates there in one of the fields bounded by other triradials. The fields and triradial rays are designated in a specific order using (letters and) numbers. The directions and lengths of the main rays of the triradii vary enormously; this naming system allows these countless variations to be classified in a specific manner and designated by short numerical formulas. (Further details can be found in the original literature — this dactyloscopy has now become an indispensable tool for criminalistics.)

The arrangement of the ridges is so individually varied that not two alike can be found amongst millions.[11] The frequency of certain patterns — spirals, arcs, etc. — appears to vary according to race, but there is no systematic regularity in this.

The respective arrangement must be governed by specific genetic units, but the relationships must be very complex. Identical twins usually have a completely identical, and often mirror-image, arrangement of the ridges, but occasionally they also have different ones, just as the same person often has different ones on the right and left. It should also be noted in particular that, from their first embryonic appearance onwards, the number of creases and their arrangement do not change.

The thickness of the skin varies extraordinarily from person to person; in terms of race, it is only known that the skin of North African desert dwellers is particularly thin and low in fat. Hottentots and Bushmen show a greater tendency for the skin to wrinkle.

In Mongolians, the sweat glands are not as densely concentrated in the armpits as they are in us.

l) Body height.

A v e comparison of people in terms of their body height not only reveals very large individual differences, but also shows that individual groups have quite different average heights. Closer examination reveals quite complex relationships, in which idiotypical and paratypical phenomena are combined. Growth normally proceeds in roughly the same way in all people; the gender differences in the growth curve – with girls overtaking boys before puberty and boys subsequently catching up and eventually overtaking them again – are the same across all races. Races that tend to be shorter generally stop growing a few years earlier than taller races; however, they are also slightly smaller than the latter from birth.

Within a race, tall individuals tend to be tall from the outset, that is, to be ahead of their peers even as small children, in

In other cases, they only outgrow them later, during puberty. This alone shows just how complex these circumstances are.

Growth depends on internal secretion. The thymus gland (?), the pituitary gland and the gonads influence growth. If one of these glands malfunctions, it can result in individual cases of gigantism or dwarfism. It is not known whether such glandular anomalies are hereditary, but it is not unlikely.

Apart from endocrine secretion, nutrition also has a very significant effect on growth. What the experiment on the T ier demonstrated—namely, that dogs or pigs from a single litter can be reared to become permanently very dissimilar siblings through very different diets (see p. 13)—also applies to humans. The height, chest circumference and head circumference of children from the poor are, on average, significantly smaller than those of young people of the same age from affluent circles.[12] These factors have probably also contributed to the clearly observable increase in the height of the European population over the last 100 years. In Scandinavia, the average height of conscripts has increased by 1½–2 cm over the last 20–30 years, and in Baden by 1.2 cm over 40 years.

However, these parakinetics are counterbalanced by idiogenic factors. It is clear to see how height is inherited within families; this has long been observed and noted even among laypeople. It is well known, for example, that King Frederick I of Prussia would only grant his tall grenadiers permission to marry if the bride in question was of similar height to the groom; he wished to ‘breed’ stately offspring
. Recent studies on cross-breeding have made it more than likely that one must assume the inheritance of ‘height’ involves dominantly inherited Mendelian factors; this is likely to be a case of polymeric inheritance. In these interracial crossings, a peculiar phenomenon occurred which can also be observed in animal and plant hybridisation, and which is referred to here as the ‘exaggerated growth’ of the hybrids. The hybrid becomes taller and stronger than both parent races.

In humans, the mixed-race offspring of tall North American Indians and North American Europeans are taller than both parent races, whilst living under the same conditions as them.

Mixed-race individuals of Boers and Hottentots are also, on average, slightly taller than their respective ancestral groups.

The origin of the so-called Pygmies, that is, 'dwarf races', has been discussed particularly frequently; the notion that they are stunted forms resulting from unfavourable nutritional conditions can be ruled out; they are idiovariations, in the formation and maintenance of which, of course, natural selection has played a role. In all 'racial heights', it is clearly evident that, despite all the factors mentioned above that influence an individual's height, the hereditary growth pattern characteristic of the race nevertheless persists, even if, in individual cases, it is not measurable and cannot be recognised as such amongst the paratypical modifications.

m) Body proportions and external body shape.

Body proportions—that is, the ratio of the lengths or sizes of the individual body segments to one another and to the whole—are so heavily influenced by external factors that any hereditary racial differences are largely masked by them. Whether, for example, the relatively long legs of Black people and similar characteristics of other races are hereditary is questionable. Irrespective of racial differences, women of all races have relatively shorter legs and a longer torso.

An increase in arm length is observed in occupations that make intensive use of the arms (blacksmiths, etc.), whilst, for example, students, civil servants, etc. have relatively longer legs, and seafarers have relatively long arms and legs. The use of the extremities thus leads to an increase in their length. Admittedly, the extent to which social selection plays a part remains open to question; the racial mix of the European population has made it particularly effective.

Human right-handedness is also worth mentioning here. It does, in fact, occur as the norm amongst 'humanity' as a whole. But it is precisely here that one sees how difficult it is to distinguish between hereditary factors and parakinetics. We do not know the origin of right- or left-handedness. In many individuals, at any rate, a preference for one hand is rooted in an anatomical basis present from birth; this is clearly evident in those who remain left-handed despite all upbringing and coercion to become right-handed.

The 'left- or right-brain dominance' underlying 'innate' right- or left-handedness is based either on directly acting genetic factors or on the position, curvature, vascular branching, etc., of the embryo.

A statistically sound study of the heredity of left-handedness is still lacking.

Left-leggedness is by no means as pronounced or prevalent as right-handedness, largely, it seems, simply because the functions of the individual legs are far less differentiated,

and where they are (jumping, etc.), habit has often been established through upbringing. —

The whole body is slightly asymmetrical; the majority of people have a curvature of the spine that is convex to the left in the thoracic region and in the opposite direction in the lumbar region; a minority have the opposite curvature. Here too, the causes are likely to lie in embryonic positioning and growth conditions.

The general shape of the body, whether slender or stocky, fat or thin, is partly determined by paratypic factors — dietary conditions, particularly during the growth period — but is in part distinctly idiotypic; metabolism and the response to specific quantities and qualities of food vary significantly from person to person.

In terms of race, that is, on the basis of hereditary predispositions, under the same dietary conditions, individual people, but also groups,
such as North African Jewish women, to accumulate fat particularly readily.

The fact that normal fat deposition on the body differs between men and women in terms of regional distribution and quantity is likely due to (hereditary) endocrine causes.

A quite distinctive form of fat deposition is found in Hottentot and Bushman women above the buttocks, causing them to protrude backwards in a hump-like manner; this is known as 'steatopygia'. Anatomically, this formation corresponds to the fat hump found in some breeds of cattle, and the fat rump and fat tail of certain breeds of sheep. Steatopygia occurs regularly and characteristically; the fat is confined to the buttocks and thighs, whilst the rest of the body may be downright lean. It is a distinctive racial characteristic that is firmly inherited and restricted to the female sex. In Hottentot-European mixed-race individuals, no true steatopygia has been observed to date, but only a strong tendency towards fat accumulation on the hips, thighs and buttocks.

As regards other details of external body shape, the difference in the female breast should be mentioned. In older women, the breast of a Black woman tends to hang down in the form of a sagging pouch; this is partly a racial (hereditary) peculiarity and partly the result of prolonged breastfeeding. (Black women occasionally breastfeed children as young as 5–6 years old, as their breasts are still functioning to feed younger siblings.)

In Bushwoman women, the breasts sit very high and towards the armpits. Among our own people, too, their position and, to some extent, their size are likely to be hereditary.

n) P h y s i o g n o m y .

What is collectively termed physiognomy varies extraordinarily from one race to another. Certain aspects of it can be clearly understood in terms of heredity. The nose varies in size and shape in all its individual parts. The overall shape of the nose is determined by its height, i.
i.e. the distance from the forehead-nose suture to the nasolabial angle, and its width measured from one nostril to the other. This is referred to as the nasal index, and a distinction is made between narrow, medium and broad forms. In narrow forms, the nostrils run as narrow slits from front to back; in the broadest forms, conversely, they run almost from right to left. The bridge of the nose may be straight, concave or convex. In the latter case, it may either curve evenly or have a sharp kink in the middle – known as an 'eagle nose' – or, finally, only the lowest section, including the tip of the nose, may be sharply curved downwards, known as a 'hawk nose'. Studies of interracial crossbreeds have shown that at least four independently inherited traits must be assumed for the nose. The bridge of the nose and the shape of the nostrils, the root of the nose and the tip of the nose can each be inherited separately. The strongly raised, convex nose is inherited dominantly over the broader, lower one. This applies both to cross-breeding between Europeans and Hottentots, and to that between Europeans and Jews. Paratypically, the overall shape of the nose is not influenced, so that observation of the nose shape is particularly important for racial diagnosis. In heredity, certain nose and facial shapes, as well as individual parts of the nose based on the aforementioned genetic factors, frequently occur together.

If these shared clusters are disrupted in interracial mixing, nose shapes emerge that appear disharmonious to us at first glance. The same applies to the entire physiognomy. The fact that the individual characteristics making up a physiognomy are each inherited separately determines whether the face exhibits breed-specific harmony or a disharmony that we perceive as unattractive. It should be clearly noted that in everyday life we often perceive a single feature in

Although we may perceive a face as unfamiliar, observation of the parents or grandparents of the individual in question teaches us that, within the racially similar features of one parent, a single trait from the other parent has been inherited, for example, a small, snub nose inherited from the mother's side in a long, narrow man's face, or a nose that is far too large and therefore unattractive, inherited from the father, in a smaller, round girl's face. Here, a vast field of opportunity for precise Mendelian genetic studies remains untapped.

Of the face as a whole, attention should also be drawn to the lips. Here, a distinctive thickening of the lower lip with a bulging protrusion of the entire chin region (prognathism) is a well-known phenomenon that appears to be inherited dominantly. It has often been observed and extensively studied in the male line of the Habsburgs. A pronounced bulging of the lip, such that the red part of both the upper and lower lips is particularly wide, is characteristic of the Negro race. For African Pygmies, on the other hand, and for some Malay groups, only thick lips—but not bulging ones—are characteristic of the race. Individual lip shapes (narrowness, shortness of the upper lip, etc.) are clearly inherited in our population. Apart from crude artificial interventions on the nostrils and nasal septum (piercings), on the upper and lower lips, earlobes, etc., we are unaware of any environmental influences on individual facial features; such influences on the face as a whole were discussed above. (p. 84.)

Conclusion.

The picture provided by the preceding investigation into so-called normal human variations is a rather complex one. Almost all forms and structures of the human body, whether considered individually or across human groups, exhibit minor and major differences. The aim was to determine which variations, taken as a whole and in themselves, or how much of such variations is hereditary, and which, or how much, is caused solely by environmental factors (i.e. peristatic). In the vast majority of cases, the hereditary component—the idiotypical component—does not manifest itself in its purest form; paratypical influences modify it. Only the idiotypical characteristics are naturally suited to characterising racial traits, i.e. traits that are hereditarily distinct from other

distinct and genealogically related groups, naturally only the idiotypical characteristics are suitable. In the case of the others, a careful analysis must identify the idiotypical component. Accordingly, the characteristics most important for a description and classification of breeds are eye colour, hair colour, followed by skin colour, Mongolian spot, hair shape, nose shape, eyelid folds, lip shape and other physiognomic details, certain mental characteristics, body height, face shape and skull shape. Of numerous other characteristics, the extent to which their formation is determined by racial inheritance and by acquired factors is not sufficiently established to allow them to be used for racial diagnosis; this applies, for example, to body proportions, many details of the skull and limb bones, muscular variations, skin ridges on the palms and soles, tooth shapes and much more. However, the former characteristics are sufficient to provide conclusive evidence that there is, in fact, a distinct, distinctly inherited and therefore essentially unchangeable differences between human beings, which divide them into groups known as races. It is only through interbreeding that these boundaries can be blurred. The task now, therefore, is to clarify these boundaries in detail; this is the task of anthropology.

2. The Origin of Races and Racial Biology.

The origin of human varieties, the so-called 'races', dates back to the very origin of humankind. In terms of its overall organisation, humankind indisputably belongs, together with all apes, to the 'primates', i.e. 'higher animals'. Within the primates, the so-called human-like apes or 'anthropoids' and humans—extinct forms and modern ones collectively termed 'hominids'—form a distinct group. Towards the end of the so-called Tertiary period, there was a rich diversification of anthropoids and hominids. Fossil finds of anthropoid apes reveal their Tertiary distribution from China and India across Europe as far as England. A major centre of dispersal—and perhaps of origin—is North-East Africa, where rich finds from the Fayum (Upper Egypt) have afforded us important insights into the evolution of apes in general. The Tertiary evolution of the ape-like primates shows that generalised forms were present in fairly large numbers at that time. Some of these have become extinct, whilst others have evolved into the so-called 'ape-like' primates of today. Of the latter—the orangutan, gorilla, chimpanzee and gibbon—each has, of course, developed in its own distinct way and adapted to specific living conditions. This entire evolution of the primate lineage also includes the line that led to modern humans. It follows naturally from the above that humans are by no means particularly closely related to any of today's great apes; rather, their lineage, together with that of these anthropoids, traces back to generalised, ancestral forms, whose organisation is in some respects better preserved in today's lower apes than in the anthropoids. Of course, the lower apes have also continued to evolve along their own lines. It follows from this relationship that we must find fossil forms which resemble modern humans more closely than the modern anthropoids do. These are then classified as hominids. The most similar is the famous Java find, the so-called *Pithecanthropus erectus*. Its geological age alone (late Pleistocene) argues against it being our ancestor. On the other hand, a very precise anatomical examination of the skull

and femurs has conclusively demonstrated that it cannot simply be classified as a human fossil, yet nor is it an anthropoid in the modern sense of the word. What we actually have before us here is precisely what the theory requires: not an intermediate form between modern humans and apes, but a generalised form close to the root, which possesses neither the characteristics of modern apes nor those of humans, but rather those from which both human and ape characteristics can be derived. It is a form that has come to a standstill on the path to becoming human, a path that also led, to some extent, towards becoming an ape.

The factors that led to the emergence of humankind were roughly as follows. The starting point was a 'primatoid' (primate-like) creature, that is, a form with climbing feet and a semi-upright posture, adapted to life in the tropical jungle. Under these conditions, a climbing creature would never have evolved into a creature with an upright gait; this could only have happened through external compulsion, that is, through the disappearance of the primeval forest. The creature had to be compelled, by secular changes in its environment, to abandon its arboreal life and adapt to the plains. The most important change in its organisation was, without a doubt, the development of the foot. It has already been said, and rightly so, that it was with the foot that man became human. Adaptation to life in the sparse parkland forest or in the bush savannah compelled the form to rise fully upright from a semi-erect posture. Such a transition from forest life to life in a treeless area can be demonstrated twice within the ape lineage. Once in the lineage leading to humans, and the other in that leading to the baboon. Here the form descended from a semi-upright posture back to a fully quadrupedal stance. The baboon is typically adapted to life on rocks; it hardly ever climbs trees. That other branch — the ancestor of humans — conversely evolved to become bipedal. The mechanical changes that the ape's foot underwent with the development of the human arched foot cannot be discussed here. Once the initial impetus had been given, and the body had been raised onto the hind limbs, all other changes to the body were a necessary consequence. For static reasons, the human spine had to develop its curves and its sharp bend at the pelvis (promontory); the pelvis had to expand; the skull could now become heavier, as it was now balanced below its centre and no longer hung obliquely at the front of the spine, as in quadrupeds. The driving force behind all this, however, was a struggle for existence that had reached its peak, and, as a consequence, the most rigorous natural selection. As long as the creature still lived in the primeval forest, adapted to it, and was optimally nourished by the nutritious tropical fruits of the forest, there was no particularly rigorous natural selection specifically for this form. But with the change in these conditions, this became all the more the case. Now, if the form was to adapt, other, new characteristics had to be bred into it. On the one hand, upon entering the open plains, a vast number of new enemies appeared—migrating plains animals already adapted to these conditions; on the other hand, the different means of obtaining food must have been particularly difficult for the creature undergoing transformation. The sparse fruits of the open steppe were no longer sufficient for sustenance. The creature shifts increasingly towards a meat-based diet; the most acute hardship sets in. Every slight improvement in the stability of its stance—which is now necessary not only at rest but also as a starting position for striking and throwing—is now vital. The intellect is now being selected for most rigorously. The mental faculties, and as their foundation

the size of the brain, are already quite large across the entire ape group, in contrast to other orders of mammals. A suitable basis for the development of a considerable brain size was therefore already in place, and it is likely no coincidence that, in Pithecanthropus, the femur already indicates an upright posture, whilst the cranial capacity has increased from a maximum of 600 ccm in great apes (today's! probably even less in their ancestors) to 1000 ccm. Only with an upright posture is such an increase in brain weight conceivable.

Now we see the first developments that we regard as uniquely human. The very beginnings of language are likely to have developed gradually at this stage, but it represented, so to speak, a tremendous leap forward when, for the first time, the species in question progressed from merely sensing the beneficial effects of fire – as apes do today – to 'stoking' it to ensure that a fire that had been kindled to provide warmth did not go out again. The art of 'fire maintenance' developed from this, and it was only then that humans rose above the rest of the animals. Now they could take control of their own body temperature and food supply. The preservative effect of fire on food (in the tropics!) and the ability to make certain plant-based foods edible in the first place through fire (certain poisonous or raw, indigestible fruits) brought enormous advantages. From this point onwards, therefore, early man exerted a strong, deliberate influence on his diet and, in the meantime, probably also on his entire life—including reproduction and the organisation of communal living—through habits that gradually took shape and became customs and laws. From this time onwards, we may increasingly regard him, biologically speaking, as a domesticated form of animal.

This entire process was so complex that we cannot imagine it having taken place repeatedly in exactly the same way and having led to a virtually identical end product. It probably occurred only o n c e. On the other hand, we may well assume that various strains of proto-anthropoids were subjected to drastic changes in their environment on several occasions; but they were unable to adapt to these changes quickly enough in every instance and became extinct. The fossil remains of anthropoids known to us today are evidence of just how severely environmental changes have decimated the anthropoid lineage. Only five forms descended from the common anthropoid-hominid ancestor have survived all those geological (climatic, etc.) changes: humans and the four great apes of today, each having adapted to its own environment

; humans have undergone the most significant changes, as they are adapted to the most drastically altered environment. We may therefore assume that the origin of humankind was a unique and unified process that took place in a single location. Of course, one should not imagine this 'location' as being too small. One would probably not be mistaken in imagining vast expanses—to give an example—roughly the size of half of Europe. An entire region of this kind, across which the primate species in question was evenly distributed, would have been subject to those long-term changes for tens of thousands of years, and the entire population of that primate species living there would have changed uniformly in the manner described. It follows from the above that we must envisage this change occurring towards the end of the Tertiary period; this is evident from geological and zoological theory, and the actual finds confirm it.

The driving factors behind this entire development were, as mentioned, changes in the environment and the resulting shift in direction and intensification of natural selection. Any slight change that was advantageous for survival, for obtaining food, for resistance to enemies, for ensuring reproduction, for maintaining health, and so on, must have given the individual in question an advantage over its competitors. This most rigorous selection transformed the generalised primate form into early humans and humans. The human remains from the Ice Age serve as genuine evidence of this gradual transformation.

They display features no longer found today, so that they are often classified as distinct species. Some of them are likely to be the direct ancestors of modern humans. One can very clearly discern successive stages of development. Geologically the oldest, and anatomically the most primitive, is the Mauer man near Heidelberg (*Homo heidelbergensis*). Chronologically and anatomically, this is followed by the Neanderthal group (*Homo primigenius*, finds from Neanderthal, Spy, Krapina, La Chapelle, Le Moustier, etc.). These forms are very distinctly different from a l l modern humans; each skull can be reliably identified as belonging to this group on the basis of its measurements. Chronologically later come forms referred to as 'transitional forms', which, however, still belong to the Ice Age: the Aurignacian race, the

(not specifically classified by race) so-called transitional forms from Brno and Brüx, etc., the finds from Oberkassel and Predmost, and then the Cro-Magnon race.

These are linked to the races of the Neolithic period—that is, geologically speaking, the present day. All these transformations are likewise brought about by the most rigorous natural selection, probably in connection with the changing conditions summarised as the 'Ice Age'. Natural selection has continuously shaped and transformed the form. Its effect on intellectual activity can be seen in the development of culture, as evidenced by thousands of finds of stone, horn and bone tools from that
'Early Stone Age', and runs entirely parallel to physical development.

It follows, therefore, that the emergence of the human species was accompanied almost simultaneously, or at least very early on, by a division into varieties. One might even say that speciation is, at the same time, the formation of races. It is entirely impossible that, within the geographically not insignificant area where this transformation took place, external factors always acted in exactly the same way, or, conversely, that the reaction of individuals and groups to these external factors was always exactly the same. Thus, slight local differences are likely to have developed from the very beginning. One may well assume that the most pronounced differences among modern humans—that is, what we today regard as the most profound racial differences—trace their origins back to the very earliest stages of human evolution itself. Thus, the three main groups—the Europoid, Negroid and Mongoloid—may perhaps be traced back to that time. However, it is in the nature of things that this is not simply a division into two or three groups, but that, from the very beginning, so to speak, secondary branches arose alongside these main branches—similar to the main branches, yet each exhibiting slight distinctive characteristics. It is therefore understandable that, for example, alongside the Negro there are certain Melanesian groups which are very close to the Negro in some respects, yet in others are more distant. This explains the futility of attempts to classify the races entirely systematically. Once humanity had reached the stage outlined above—that is, in possession of a primitive culture, and above all in possession of fire (a long period of fire preservation is followed only by the period of the art of fire-making)—in other words, as shown above,

human beings were now to be regarded, biologically speaking, as domesticated ( ), phenomena began to emerge which we can observe today in domesticated animals. New forms and racial differences arise, after all, through the occurrence of idiovariations, and it seems that in the state of domestication, idiovariations occur particularly frequently and, above all, are more frequently preserved through selective breeding. All the characteristics found in humans as racial differences also occur in domesticated animals, and conversely, almost all the characteristics of our domesticated animals are also found in humans. Above all, one should recall the variation in hair in terms of shape, length and colour, as well as the variation in skin and eye colour, body size and nose shape. As an example, one might point to the emergence of so-called fair skin and blond hair. It can easily be demonstrated that individuals with pale pigmentation occasionally occur among all dark-skinned races. Under natural conditions, however, fair skin could not survive in the tropics and was weeded out. The tropical sun damages the unpigmented body to such an extent that the emergence of a fair-skinned and fair-haired race is only possible in cooler climates. The fact that the fair-haired Northern European exhibits the exact same pattern of pigment distribution—particularly in the iris—as light-coloured domesticated animals, but quite differently from polar animals, is clear evidence that this is a form of domestication which was able to persist in the cool climate, whilst similar idiovariants in the tropics were eliminated whenever they arose or continue to arise. Natural selection processes continue to exert a constant influence on modern humans as well, particularly those with little cultural development. People living in the most primitive of today's cultures remain very much subject to natural selection, particularly with regard to their ability to obtain food, to endure long marches, heat, thirst, hunger, to withstand blood loss, to make do with hard-to-digest food, to give birth easily, to breastfeed, to be in possession of healthy limbs, to have keen and normal senses, and to possess sufficient mental faculties. The undoubtedly frequent periods of drought and famine resulting from the death of game periodically intensify such selection processes to the extreme. Thus, constant care is taken to ensure that the race remains at its peak. It is only semi-civilised and fully civilised societies that restrict this selection—as will be shown in the third section—partly consciously, but above all unconsciously, to a greater or lesser extent. In

these natural selection processes, which have evidently bred many of the existing racial differences, sexual selection is now added. This is likely to have bred characteristics such as beard shapes, the Hottentot tail and others.

The origin of the races just described now also sheds all manner of light on that field which we may summarise as racial biology. All the races of the earth have interbred with one another in manifold ways and in many places. The problem of interracial crossbreeding has only become comprehensible to us since the discovery of Mendel's laws of segregation. And it can be demonstrated that all interracial crossbreeding is governed by these laws. We shall not retrace the interbreeding of individual traits here, but merely draw attention to a few more general phenomena. Firstly, it has been established that interbreeding between all races, and likewise between hybrids, is fertile. Fertility also appears to decline only slightly; at the very least, cases of reduced fertility among mulattoes can be explained by other factors. It seems particularly important to note that the precise observation of interracial crossbreeding, carried out in the light of Mendel's laws, has taught us that there is no such thing as the 'prepotence' in the inheritance of individual races that was previously generally assumed. The assumption that so-called 'wild' races – and here one has also thought of Black people – always prevail over so-called 'civilised' races in crossbreeding is certainly incorrect. It is by no means a question of the predominance of any single race; rather, in humans—just as in the thousands of animal and plant experiments—individual traits are inherited as either dominant or recessive. If a race possesses many dominant traits, it will simply appear more prevalent in the hybrid population than the other parent race, which has more recessive traits. This readily explains why, when Blacks and Europeans interbreed, the mixed-race offspring are generally dark-skinned and dark-haired. The view that individual races 'come through' as such is then further supported by a psychological fallacy, particularly when it comes to the interbreeding of European races with Jews. In the F_2 and

subsequent generations, one very frequently finds individual 'Jewish' traits reappearing, even in cases where the F_1generation may not have looked Jewish at all; yet one overlooks all those cases in which, conversely, only non-Jewish traits are observable – cases in which one would, logically, have to speak of the non-Jewish component manifesting itself. Precise studies of intermarriages with Jews have shown that here too, as is entirely to be expected, only individual dominant traits come to the fore—namely black hair, a convex nose, and perhaps one or two other features of physiognomy. Conversely, in intermarriages between broad-nosed Eastern European Jews and Northern Europeans with sharp, narrow noses, this Nordic nose is clearly dominant.

The emergence of new races can never occur through interbreeding alone. Interbreeding can only create new combinations, without the old characteristics disappearing as a result of the interbreeding alone. The disappearance of the old and the genuine creation of the new can only be achieved through selection. The new combinations can therefore be selected and culled in such a way that all individuals possessing certain characteristics disappear, and all individuals exhibit certain new combinations. A new race has then arisen as a result of interbreeding; the factors bringing this about were selection and culling themselves.

The most intense intermingling of races occurs when entire peoples intermingle; it is here that the closest interrelationships between 'people' and 'race' exist. The terms themselves have nothing to do with one another in and of themselves. The term 'people' refers to a group of people united by shared cultural assets, the most important of which is language, whilst races are groups of people who share the same genetic makeup. Nevertheless, the concept of race is of the utmost significance for the concept of a people. The bearers of the relevant national culture are, of course, individuals of a particular race or a particular racial mixture. The consequences of a mixture of two peoples—which usually occurs when one defeats the other by force of arms and imposes its own national identity upon it, though occasionally also takes the form of a peaceful and gradual assimilation—are of a quite unique and profound significance for the racial composition of all present-day peoples. The clearest example is probably the case where one people, as

conquering class over another. In doing so, it may be racially related to the other, or it may have an entirely different composition. The language and other aspects of culture may also be closely related between the two, or, conversely, entirely dissimilar. The subsequent course of events can vary greatly from case to case. Figuratively speaking, the cultural elements engage in a veritable struggle for existence with one another. The language of the conquering people may completely destroy and supplant that of the subjugated, taking its place, though not without itself absorbing and retaining foreign elements and undergoing certain other changes in the process. Consequently, a completely different language may later exist in the region in question than was previously the case. Thus, Indo-European languages are spoken today across large parts of Europe, where other (non-Indo-European) languages are historically documented to have once prevailed. The same applies to other cultural assets, both intellectual and material. The bearers of the new language may, for their part, initially maintain a relatively 'pure-blooded' distinction from the subjugated peoples for a long time and continue to reproduce. This purity is most pronounced when barriers of habit, custom and law—erected out of racial pride—separate the victors from the vanquished, as we see in its most extreme form in ancient India, where supposedly insurmountable castes divided the various social strata. It is highly significant that the Indian word for 'caste' means 'colour', by which the conqueror proudly distinguished himself from the ancient, defeated people. But even where religiously entrenched caste boundaries were established, and even more so where only simple laws and prohibitions on marriage—or even merely custom—prevented a more intensive intermingling between the two groups, there was never any real separation. Slowly or quickly, drop by drop or in torrents, the blood of one component mingled with that of the other. And now peculiar processes of natural selection set in. If the conqueror arriving from outside was not at all adapted to the new climatic and other conditions—that is, if he came from entirely different regions—then purely natural selection processes alone will bring about a very strong culling of those individuals who manifest the relevant traits as carriers of the relevant genes. Added to this are specific processes of selection determined by social factors. In almost all civilised peoples—and in the West, without exception—we observe a considerably lower birth rate among the socially superior classes. Furthermore, there is a

elimination of capable male individuals through wars, political persecution and the like. If all this occurs repeatedly, generation after generation, the bearers of these hereditary lines are bound, sooner or later, to be completely and utterly eradicated from the mixed population in question. Long before anything was known of such processes of selection, before the peculiar laws of heredity were clear to us, it was observed that, following periods of such mixing between two peoples, the one ancient, indigenous type reappears almost in its pure form. People have spoken of the 'separation of the races'; this is not a specific hereditary process, nor an inexplicable resurgence of the old, indigenous race, but merely the elimination of the maladjusted immigrant elements, brought about by natural and social selection. The end result is therefore a new people with a new language and other cultural assets, but of the same old type that the original people had previously possessed; only isolated traits—which, due to the laws of segregation as a consequence of individual hereditary factors, recur time and again here and there—reveal, even in terms of physical racial characteristics, that a different foreign influence had once been at work. In the cultural sphere, on the other hand, it is that episode of mixing that has remained the decisive factor, and the old has been lost save for a few remnants.

Admittedly, this need not always be the case. The reverse situation also occurs, whereby the conqueror, when encountering favourable biological conditions, in turn almost entirely displaces the subjugated people and, having chanced upon conditions to which he is adapted, survives without difficulty, even in racial terms. In such cases, too, he might adopt certain intellectual elements—such as settlement patterns or cultural forms—from the subjugated and subsequently eradicated people.

However, a people's racial composition, alongside other factors, has a decisive influence on its fate and on its cultural and intellectual capacity under all circumstances. It should not be overlooked that a host of external factors—geographical location, relations with neighbours, their nature and achievements, and a thousand other things besides, which history usually presents as the sole determinants—are certainly of decisive importance for the rise or fall of a people throughout history. But alongside these,

undoubtedly, racial endowment plays an immense role. Just as in physical terms, so too in intellectual respects are the individual human races extraordinarily diverse. Imagination, drive, intelligence, etc., have been bestowed upon the individual races to extraordinarily varying degrees and in the most diverse combinations, being hereditary and inalienable. Just as the environment influences physical characteristics in a paratypical manner, so too—as is the case with the individual—will the circumstances of life and destiny, in a people composed of thousands of individuals with their racial predispositions, modify the expression of mental hereditary traits in manifold ways. But the predispositions themselves are a given, and since peoples differ racially, their mental aptitudes also differ. Generation after generation, the North American Indians sat upon copper deposits; they lacked the intelligence to invent the technical processing, smelting and casting of copper, let alone the production of bronze. Many of the various peoples of the South Seas are probably of the same age; yet some have not progressed beyond the most primitive stone-age culture, beyond the stage of hunter-gatherers, whilst others have created a quite remarkable slash-and-burn agriculture. And the peoples of the Near East and Europe progressed to plough cultivation and the entire civilisation that followed from it; they are no older than their neighbouring African peoples, merely different in their intellectual capacities. It is becoming increasingly clear that the racial factor—that is, specifically the inherited intellectual aptitudes—is one of the decisive factors in the entire course of a people's history, alongside other factors, as must be emphasised time and again. And it is precisely here that the significance of racial mixing becomes most evident. In a great many cases, the conquering class will assume the actual leadership; the fact that so often, following a period of intensive racial mixing—that is, when the intrusion of a conquering class has led to the formation of a new people from an old one, as in the case of the Indo-European peoples, the ancient Greeks, the Latins, and so on— a particular intellectual upswing, a flourishing of art and knowledge, and a political heyday are observed is no coincidence, but a process determined by the laws of heredity; new 'mix-variations' also arise in the intellectual sphere. The prerequisite here is that the newcomer and the subjugated people are not too far apart in their intellectual aptitudes, that they

'fit' together. It will never be possible to say in advance whether a cross-breeding will prove favourable or unfavourable in this respect. It will only be possible to assess this to some extent each time after the process has been completed. The economic, artistic and political flourishing of a people will depend on its possessing, at all times, a sufficient number of those aptitudes necessary for the bearers of the relevant achievements, and, even more so, that it possesses at all times a small number of gifted individuals who can serve as leaders and guides in the economic, artistic and political spheres. If spiritual leaders are lacking, a nation will be able to endure this deficiency without obvious harm only as long as no external danger threatens; but when danger itself arises, it will certainly succumb. Thus, these anthropological questions play a decisive role in the fate of a people. A people may indeed grow old, but a race never does. Human races do not, in themselves, grow old or die any more than animal or plant species do. They can only be wiped out, that is, meet an unnatural death. What the historian regards as the degeneration, decline and ageing of a people—indeed, what he sees as the downfall of a people—are the consequences of misguided selection among the racial components of the people in question. If the hereditary bearers of the relevant endowments, if the racial leaders, are eradicated or eliminated through any process of selection, then the people are simply no longer the same as they were before. When a people has become a different one in its racial composition—whether through warfare or through peaceful, gradual racial mixing brought about by immigration—its intellectual achievements too must inevitably change. This change—manifested above all in economic and political decline—is, as mentioned, frequently interpreted by historians as a sign of ageing; in reality, it is a racial transformation. The Greek people in the time of Pericles were quite different from the Greeks who trembled before the Roman emperors, and quite different from the inhabitants of modern-day Greece. In this context, one can only briefly allude to the fate and nature of our own people over the course of the centuries; indeed, the fate currently being experienced by the German people also has its anthropological cause.

3. Racial description (anthropography).

The systematic description of the various human races is known as anthropography. It describes the individual groups according to their anthropological characteristics, compares them and attempts to classify them systematically on that basis. The commonly used classifications are rather outdated and worthless.

> Naturally, any classification that takes into account factors such as language alongside physical characteristics must be rejected on principle. As demonstrated above, language and folk culture can change even without a corresponding change in race. A Portuguese person speaking an Indo-European language is not, therefore, racially related to a Swede simply because the latter also speaks an Indo-European language. The Black people of North America have lost all trace of their Bantu languages and speak English; racially, they are of course still Black. For this very reason, the naming of races must in turn avoid names that are applied to peoples or even languages. The most consistent approach would be to use names based purely on physical characteristics, as is customary in zoology and botany. However, there is nothing wrong with names based on geographical distribution either; they are perhaps even more appropriate for humans than the former. A detailed account of the various classification attempts need not be given here.

The following anthropographic account takes a broadly geographical approach, whilst certain larger anthropographic groups are to be singled out , taking into account specific racial interrelationships. The non-European races can only be described in very broad terms — space constraints preclude anything more.

E u r o p e .

Despite the countless prehistoric and historical mass migrations that have taken place in and across Europe, it can be observed that certain pockets of individual races have persisted in specific locations. If one plots the distribution of the most important anthropological characteristics on geographical maps of Europe, the combination of these characteristics clearly reveals that the geographical distribution of certain races

is verifiable. It goes without saying that the so-called borders of Europe are not racial boundaries; one of the European races extends from south-eastern Europe far into the Near East, whilst the extent of a second race from Central Europe into Asia must be dealt with separately later on.

The so-called 'Nordic ra ce' occupies the north and north-west of Europe, extending in Central Europe as far as the Alps, and in particular the coastal regions of the North Sea and the Baltic Sea. It forms the most compact block in certain parts of Sweden, as well as in Scotland; elsewhere in the aforementioned regions, it is mixed to a greater or lesser extent with other races; finally, it is also present as a minor component in mixed populations in Eastern and Southern Europe. In North America and other overseas territories, it is found in accordance with the origin of the immigrant populations concerned. The characteristics of the Nordic race are as follows: the individuals are tall; the average height for men is 1.73 m, which is associated with long limbs. The skull is long and narrow, with the occiput projecting strongly and being highly arched. The face is also long and narrow; the nose is prominent, narrow, with a high bridge, sometimes straight, sometimes with a small bump at the junction of the cartilage and bone. The chin protrudes distinctly; the cheeks are narrow and set back; the plain, soft hair, which tends to curl, is light in colour – blond or reddish – and the eyes are light, ranging from blue to grey. The skin is fair, tanning scarcely at all in the sun and, indeed, sensitive to its effects. Within the race, individual sub-types of slight local variation can be identified, which cannot be discussed in detail here.

The 'Mediterranean' race is found in the coastal regions of the Mediterranean Sea. Spain and Portugal, the small and large Mediterranean islands, and southern Italy as far north as Rome, are home to a compact group of this race. Elements of it are found on the southern edge of the Balkan Peninsula and on Crete. Furthermore, the entire northern edge of Africa, from Egypt to Morocco, is populated by this race (though in a highly mixed form, see below), and from there waves extend inland: on the eastern side, upstream along the Nile; on the western side, along the coast, certainly encompassing the West African islands as well, and everywhere here in a highly mixed state. The race is of small stature, the smallest in Europe. Men measure on average 1.61
m. The race has a narrow, long skull, like the Nordic race, but smaller

and with a steeper forehead. The face is narrow and oval, the nose straight and slightly prominent, but smaller and somewhat broader than that of the Nordic race; the angle between the forehead and the nose is often particularly flat (Greek profile). The hair is very dark brown, described by laypeople as black. The eyes are also dark brown. The skin has a distinct brownish tone, which deepens to an even, light brown when exposed to the sun.

The 'Alpine' race inhabits a broad strip stretching from east to west across Central Europe within the Alpine region. The Alpine race is found in its purest form in south-western and central France, from Gascony through Auvergne to Franche-Comté, then in the Alpine region proper, particularly on its southern side, before spreading from the Alpine massif in isolated bands northwards into the territory of the Nordic race and southwards into that of the Mediterranean race, extending clearly as far as central Italy. From Tyrol and the Eastern Alps, an area of the Alpine race—in a more mixed form—extends northwards and north-eastwards beyond Central Germany; its eastern boundary cannot be defined (due to a lack of anthropological data). Similarly, it is still impossible today to specify a boundary to the south-east against the territory of the Dinaric race. The characteristic features of this Alpine race are as follows. Stature is of average height, with the average for men being 1.63–1.64 m; the skull is round, with an average cranial index in living individuals of 85 to 87; the forehead rises straight upwards; the forehead and parietal protuberances are somewhat pronounced; the occiput is evenly arched, without protruding particularly. The face is broad and rounded, the chin pointed; the nose is coarser, shorter and broader than in the other races, and slightly concave. Hair and eye colour are medium brown; the skin colour is not as light as in the Nordic race, but lighter than in the Mediterranean race, tanning evenly in the sun.

Finally, the 'Dinaric' race.[C] Its main stronghold is the Balkan countries, perhaps most concentrated in Bosnia and Herzegovina, followed by Croatia, Slavonia and Albania. It extends as far as the southern tip of the Balkan Peninsula, although the Mediterranean race is also widely distributed in Morea. To the south-east, the race can be traced across the strait to Asia Minor, where it cannot yet be distinguished from the 'Armenoid' race (see below). To the north-west, it extends into the Alpine region, and the Dinaric element is likely to reach far into southern, and indeed central, Germany via the Austrian Alpine regions. The type is unmistakable.

The hair and eyebrows are black-brown, the hair is straight, and beard growth is profuse. The head is short and high, not overly broad, so that the index averages between 81 and 86. A particularly characteristic feature is the exceptionally flat contour of the occiput, so that the head appears as if cut off at the back when viewed in profile. The face is long, coarsely modelled and features a particularly large, strongly protruding, curved nose, often with a bold sweep. No other race has a larger one. Aquiline noses and strongly curved hawk-like noses are represented in roughly equal measure. Finally, the average height for men is 1.68 m to 1.72 m, which is very considerable.

These four racial types, in intense admixture, make up the population of Europe. Added to these are external foreign elements, which intermingle with the population to a greater or lesser extent. From the east, above all, the Mongoloid race; in the south-west, Arab influences; and throughout Europe, the Jewish population.

First, however, the question arises as to the origin of the racial profile described above. Even today, no direct link can be established with the oldest European types (Neanderthals, etc.). To some extent, however, the idea that the Cro-Magnon race, known from the end of the Palaeolithic period, forms the starting point is satisfactory. The Mediterranean and Nordic races are likely to have developed from this. Whether it arose from the (as yet unsubstantiated by finds) ancestor of the Mediterranean race — it is, above all, much more robust than the latter — or whether, conversely, the present-day Mediterranean form evolved from it through modification, remains an open question. (There are clear connections between the Cro-Magnon skull and skulls from Tenerife and Egypt.)

In any case, we may assume that the Mediterranean race had occupied roughly the area of its present-day distribution by the end of the Palaeolithic period. On the other hand, from an anatomical and racial perspective, there are connections between the Cro-Magnon skeleton and the forms revealed to us by the skeletal remains of the so-called megalithic population, that is, the builders of dolmens (giant stones, burial chambers) from the Neolithic period along the Scandinavian, Danish and other coasts. These, too, are likely to be derived from the Cro-Magnon forms. A certain degree of development and change must surely have taken place. One may therefore assume that, as the ice that had completely covered the northernmost parts of Europe receded, humans followed the reindeer herds as they migrated northwards, and that in the process a group, becoming isolated there, evolved into the Nordic race. One might perhaps assume a rather small group which, under the drastically changing conditions (climate, game animals!), acquired all the distinctive characteristics through renewed, intense natural selection and inbreeding – characteristics which, within the whole of humanity, are unique to the Nordic race. Since, due to the ice, there can certainly not have been any population here previously, this Nordic race must therefore be regarded as the most original and as having originated in this very place. Relatively early on, a second wave of immigration arrived from the east, comprising the ancestors of today's Sami. From the very beginning, alongside the long, narrow skulls of the megalithic skeletons and those of all subsequent prehistoric periods, one finds a mixture of a few round skulls, right up to the present day, where, alongside the long Swedish skulls,

we can observe the round Lapp skulls. And on the southern edge of the original range of the Nordic race, the Alpine man probably intermingled as early as prehistoric times; we can identify his skulls as the so-called Borreby type in Denmark and also in the Netherlands. To the east, the Mongoloids were probably already present at that time. And the question arises as to whether the Alpine race was not originally simply the western branch of the same group. There is considerable evidence to support this, but it would probably be incorrect to put it in such terms as to say that the Mongoloids advanced westwards during the Early Stone Age as far as the Atlantic Ocean; rather, it would be more accurate to describe the situation as follows: we may assume that, at the very moment the modern Mongoloids were forming—even before they had developed their typical characteristics and distinctive features—a western group branched off. Consequently, this group possesses certain Mongoloid characteristics (skull shape), but not yet the shape of the eyelids and eye slits, the Mongolian spot and other features. The north-western branch, on the other hand—the Lapps—already possessed those characteristics; they therefore probably only migrated after the Mongoloids had truly become a distinct race. Those early Alpine peoples, who arose from the as yet not fully specialised Mongoloid stock, thus inhabited Europe as early as the Ice Age; they advanced northwards as far as the Jutland Peninsula, north-westwards, as mentioned, as far as Holland—perhaps even crossing over to England—and southwards we still find clear traces of them in Spain.

Finally, as far as we know, the Dinaric race did not arrive in Europe until much later. There is no evidence of it during the Ice Age. It is reasonable to assume that at that time it was confined to the Near East. We find the first traces of it only in the Neolithic period, and it is likely that it spread considerably once again during the Bronze Age. In the Neolithic period, the outermost outposts are thought to have left their remains in the so-called round barrows of England. It is therefore no surprise that we still find Dinaric racial characteristics today right into the heart of Europe. Similarly, such Armenoid-Dinaric elements appear to have reached from the Near East and south-eastern Europe, via Crete to the west, as far as Sicily, and southwards to Egypt.

During the Early Stone Age, the Black population of Africa had also temporarily expanded as far as the southern edge of Europe, as evidenced by the 'Grimaldi find' (near Menton); however, it did not make a significant contribution to the later population.

Once the origins of the races have been established in this way, it is necessary to consider briefly the events that took place between those earliest beginnings and the present-day situation. If, at the end of the Ice Age, the three largest European races lived roughly in their present-day habitats, evidence of numerous migrations allows us to draw a great deal of information, even regarding the proportions of admixture amongst today's peoples. What is referred to in history as the 'Great Migration' is certainly only the final – and arguably not the largest – phase of massive migrations that are becoming increasingly evident from an anthropological and prehistoric perspective: Prior to the Germanic migrations came the Celtic and Slavic migrations, and before these, the prehistoric migrations which, during the Neolithic period, brought about large-scale population shifts criss-crossing the whole of Europe. It is not yet possible to reconcile cultural spheres with anthropological types everywhere, but in

in certain areas, racial shifts clearly corresponding to these population movements can be observed. In the eastern part of Central Europe, for example in Bohemia and Silesia, people with round skulls (Alpinus?) were the bearers of the so-called 'Linear Pottery' culture of the Neolithic period, and one can trace how the 'Corded Ware' population, which is dated to a later period, displaced them. The latter group had long skulls, and in the graves the male skulls are predominantly long-skulled, whilst among the women there are many round-skulled ones—the typical picture of a settled population being overlaid by a conquering invading group. Within the same Neolithic period, the pile-dwellers of Central Europe were of the Alpine race, and around the same time we find a nomadic, migratory population whose cultural artefacts are characterised by the so-called bell beakers, with the distinctive skull shapes of the Dinaric race. Here, too, therefore, there were migrations and waves of settlement. And it is likely that the development of the later Bronze Age and Hallstatt period was also shaped by such shifts. The shifts during the Celtic and Germanic periods become much clearer in the skeletal finds, where typical Celtic, Germanic and Slavic graves reveal the remains of Europe's inhabitants around the beginning of our era. The skull type found in the so-called 'row graves' of the Germanic peoples of the Migration Period – that is, the Franks, Saxons, Alemanni and all the others – is entirely identical to the pure Nordic racial type as found in Sweden, regardless of whether it is discovered in southern Germany, France or Italy in the relevant Germanic graves.

Closely linked to these prehistoric population movements is the question of the original homeland of the Indo-Europeans. All research in the fields of linguistics, archaeology and anthropology unanimously suggests that the cradle of the Indo-European languages is to be found in Northern Europe. Whether this refers specifically to Scandinavia, the North German Plain or the Baltic region is irrelevant here. However, as shown above, the Nordic race had been established there as the original race from the time when the retreating ice first made human settlement possible at all. It is the bearer of the Proto-Indo-European language and culture, and wherever Indo-European languages were subsequently carried as they dispersed into the individual tribes

, the bearers were individuals of the Nordic race, or a mixture of this with another. The last great waves that emanated from the ancestral homeland—the Celts, Germanic peoples and Slavs—were originally not racially distinct. Their fates, however, subsequently diverged greatly. The Celtic wave, insofar as it remained in Europe, spread partly into the territory of the Mediterranean race (Spain, Italy), partly into that of the Alpine race (Central Europe, France, Southern Germany, Switzerland, Northern Italy, etc.) or into the territory of the Nordic race (England), and finally, in part, into the territory of the Dinaric race (the Balkans). In the first two regions mentioned, the race became darker through processes of selective intermingling, as described above; that is to say, in Central Europe it mixed with the Alpine race, whilst in Southern Europe it was absorbed by the Mediterranean race. The culture, however, was preserved; the new bearers of this Celtic culture in Central and Western Europe were now, for the most part, dark-haired, and the Germanic peoples who arrived later found quite a large number of dark-haired 'Celts' in southern France, for example. And even today, the layman among us largely imagines the Celts to be dark-haired and attributes the present-day dark-haired elements in our population to the Celts, whilst in reality they trace back to the Alpine race of the Late, indeed Early, Stone Age. The eastern stream, the Slavic one, was the most severely diluted in racial terms. Its task was to preserve Germanic culture from the Mongolian tide – a role that may seem insignificant in the grand scheme of world history, but which was in reality of extraordinary importance. Like a living wall, the originally tall, fair-haired Slavic people pushed eastwards ahead of their Germanic brethren and absorbed the Mongolian wave, wave after wave. And only once they had, so to speak, been filtered, tempered and blended did the Mongolian racial elements later reach the very heart of Europe – as far as and beyond the Elbe, into the Germanic world – as a result of the east-west Slavic migrations. Admittedly, we find today individual Mongolian traits as an influence within the Nordic-Alpine mixture of the German people, but this would have occurred on an extraordinarily larger scale—and with a far greater impact on the whole—had the Slavic barrier not held back the main force. What many people describe as the 'Slavic type'—broad faces, somewhat prominent cheekbones, stocky builds—is in reality a Nordic-Mongolian mixture. Today's Slavs, without exception, have a strong Mongolian influence; the southern Slavs are mixed with the

Alpine and Dinaric races. Finally, the Germanic stream remained closest to its homeland and was least influenced by foreign races. In southern Germany there is a strong admixture of the Alpine race; in Scandinavia, Mongolian influences from the Lapps; and in north-eastern Germany, the influences just mentioned from the (later) Slavs. In north-western Germany, the Netherlands, Belgium and northern France, the Nordic race predominates over vast areas. In the latter two, there has been a gradual admixture of the Alpine race. Where the Germanic wave ebbed away and moved even further from its original homeland, it died out. One need only recall the Burgundians, Goths, Vandals and others. Yet even today, the southern European countries have, numerically speaking, more fair-haired people than, for example, Scandinavia has dark-brown-haired people, even though the dark trait is dominant in heredity; as a result, many dark-haired southern Europeans mask the fair-haired trait, whereas no fair-haired northern European harbours the dark trait within themselves; a clear sign of the strong racial influence on Southern Europe exerted by the immigration of the Nordic race. In Great Britain, on the southern periphery, there was a very significant admixture of the Mediterranean race with the Nordic race, which, incidentally, predominates there. The admixture of the Alpine and Dinaric races is likely to be minimal.

Thus, all the peoples of Europe today are composed of the same races; what distinguishes them, however, is the number of components, their relative proportions, and the extent of foreign influences. Of these, the Mongolian influence has already been mentioned; furthermore, in the case of the Spanish population, attention should be drawn to the strong influence left by the Arab invasion and rule, comprising partly the Oriental race (see below) and partly Negroid elements brought with it. The other historical waves of immigration also continue to have an impact today: the settlement of the Mongolian Magyars in Hungary, of the Mongolian Bulgarians and Turks in the Balkans, the Hunnic invasions, and so on. Finally, it should be noted that, in thousands of individual cases—given the freedom of movement that prevails today and has prevailed in this regard throughout the past centuries—the intermingling of individuals of foreign races has taken place continuously. One must also bear in mind the individual Negroes, Mongols, etc., who were imported. Furthermore, individual Southern Europeans migrate to Central and Northern Europe and vice versa; the most significant role, however, is played at present by the Jewish influence, which

brings with it a considerable number of Near Eastern racial elements (see below). All this accounts for the often peculiar traits found in individual families and people, which can be explained by the laws of heredity. The influx into Germany of hundreds of thousands of prisoners of war from all corners of the globe, the non-white garrisons in the currently occupied territories, and above all those in France over the past six years, will become clearly evident in the future! There can be no doubt whatsoever that the intellectual differences between the individual European peoples also depend on these racial differences. The four major European races and the non-European races that have intermingled with them naturally each have their own distinctive genetic makeup, and the intermingling and selection that have taken place within a people will have had a corresponding effect and have determined the psychological disposition of that people. Nor should we regard this process as complete even today; before our very eyes, natural selection is certainly still taking place—including with regard to the various racial components of a people—with varying intensity, just as it has throughout the past centuries, indeed millennia; and so, even today, the composition of every single people is changing through this ongoing process, thereby determining, amongst other things, its fate.

Finally, we should also consider the European population of the overseas countries, particularly the United States of North America. The same patterns of intermingling that we see in Europe are also evident there. In the individual South and Central American states, there is a very strong influence of African blood, which can, in part, be clearly traced in cultural development. It is, of course, of considerable significance for the racial composition of the North American people that, in the past, the main source of its immigration was first England and then Germany, whilst in recent decades Southern and Eastern Europe have supplied its population in ever-increasing numbers. Despite the social differences between the populations from these various sources—which still largely persist today—they will intermingle in the future. In this process, social (and natural?) selection processes — above all, varying rates of reproduction — will play a significant role, so that it is probably impossible today to predict how this population will develop; it will not simply be a matter of calculation based on the number of

immigrants! The fact that even immigrant 'Germans' or 'Italians' can, in racial terms, be very different from one an , hardly needs to be emphasised again. —

The same applies to the other major European settlement areas across the seas, such as Australia, South Africa, etc.

In light of the above, this racial shift in immigration will also be of significance for the future history of the country.

> Here, we shall briefly touch upon the situation in India. In the first half of the second millennium BC, the 'Aryan' Hindus lived as pastoral peoples in the Punjab. Around 1400 BC, they advanced, driving the indigenous Dravidian population southwards and gradually merging with them to form a single people, into which elements of a more primitive indigenous population (the Veddai race, see below) were also incorporated. The language and culture of the Hindus are Indo-European. Stature, skull shape, nose shape and skin colour betray the Nordic race; admittedly, the skin is no longer 'white'; the mixture is so intense, and in particular the selection—or, as a result, the elimination—of the fairest-skinned individuals, who were entirely unprotected against the tropical sun, has been so effective that the purely homozygous fair-skinned individuals have been and are being eradicated. The highest castes (the Brahmins) display these characteristics most clearly. It has been said, and rightly so, that amongst this Indian people, an individual's social standing is inversely proportional to the width of their nose! It is precisely here in India that one can see how racial mixing and natural selection operate, how nose shapes, skull length and body height have been preserved, whilst the fair-skinned traits have been weeded out. Thus, only a few characteristics of the Nordic race have survived here — along with the magnificent cultural and artistic legacies created by the people who were its bearers.

Pre-Asian.

Prehistoric and anthropological research makes it seem fairly certain that, up until around the second millennium BC, a reasonably uniform population inhabited the whole of Asia Minor, Armenia and Mesopotamia, which we may describe as the 'Armenoid' race. It is extremely closely related to the 'Dinaric' race described above; the only difference is a slightly smaller stature. The two are likely to be sister races. We still find them today in relatively pure form in the areas mentioned, where they were able to preserve themselves, sheltered from the upheavals of the subsequent migration of peoples. For instance, amongst the remote mountain peoples of the Near East—such as the so-called Allevi, Kyssilbasch, Tachtadschi and others—and then in the Armenian Highlands, amongst noble families, in sects and the like. This powerful population, which by around the middle of the second millennium BC had already formed peoples and empires in its own right

, the most significant of which was the Hittite state, was racially influenced from two directions. From the north-west, Indo-European hordes invaded via the strait into Asia Minor, bringing with them at least a strong element of the Nordic race. The Thracians of the Greek authors and the Amorites of the Bible belong to this group, albeit from a later period. The former were the earliest precursors. But the influence that came from the south was far more profound. In the first half of the second millennium, Semitic migratory hordes advanced up the Euphrates and Tigris. The biblical legend tells us of this. As a result, large sections of this population—composed of Armenoid racial elements—were culturally Semitised; Semitic peoples and states emerged; physically, they were transformed into a mixed race. The newcomers themselves, however, brought with them, as a race, what
is to be called the 'Oriental' race. The original homeland of the Oriental race was probably the Arabian Peninsula during the diluvial period, where we must imagine it—just as we do the present-day Sahara—not as a desert but, as a result of the period of rainfall, as a fertile region, well-suited to serving as a site for the distinct development of a race. The Oriental race is characterised by a small stature, a narrow, long skull, a narrow face with somewhat fleshy, thick lips, and a narrow, prominent, evenly curved nose that is not overly large. One might picture the faces of refined Southern European Jews, in contrast to the coarse features of the large-nosed Armenoids. The Oriental race is likely to be very closely related to the Mediterranean race; both are branches of the same root. Hair and eye colour are dark brown, whereas the skin seems to have remained or become particularly fair. This, then, was the racial type of the original Semitic immigrants. From the mixture of the Oriental and Armenoid races as the foundation, the actual Semitic peoples—the Assyrians, Babylonians, Hebrews and others—then emerged. But just as foreign influences can be detected in the main races of Europe, so too is the case here. Firstly, the Nordic component already mentioned; secondly, quite clearly a Negroid component—albeit not strongly represented—which must have spread across from the north-eastern corner of Africa in exactly the same way as it did across the southern edge of Europe (cf. the Grimaldi race mentioned above). Finally, in Sumerian art we find depictions of individuals with small, pointed noses; either an Alpine influence can still be assumed here (or a south-western Asian

Mongoloids?) or, which is far more likely, a branch of the Mediterranean race, which, as will be shown shortly, was firmly established along the entire northern edge of Africa.

If, for reasons of particular interest, the Hebrews are to be singled out once more from among these Semitic peoples, the racial mixture just mentioned naturally applies to them as well; they consist of the Armenoid and Oriental races as their foundation. When we distinguish today between two branches of Judaism—the Sephardim and the Ashkenazim—the former is characterised mainly by a predominance of the Oriental race, whilst the latter is characterised more by the Armenoid race. The differences among Jews in Europe can be explained by intermingling with the respective peoples amongst whom they live. Southern Jews have absorbed a great deal of Mediterranean blood, whilst Eastern Jews have absorbed Alpine and Mongoloid blood. What we are seeing is racial mixing. In light of the above, it is almost superfluous to emphasise once again that one can speak of a Jewish race any less than of a Germanic race; yet it goes without saying that both the Jews and the Germanic peoples represent distinct racial mixtures . One can therefore quite rightly speak of the racial characteristics and races of the Jews and the Germanic peoples and distinguish between them sharply and clearly.

Egypt and North Africa.

As regards the anthropology of Egypt, the circumstances are particularly favourable, as a complete record of cranial material spanning more than five millennia is available. From the earliest prehistoric finds onwards, we can identify two main types within Egypt's population. One is easily recognisable. It is a distinct Negroid population, which at times extended as far south as Lower Egypt. Even during the heyday of the Egyptian Empire, it constituted a quite significant component of the Egyptian people; and as the grave goods demonstrate, it was by no means merely a servile population, but Negroid individuals were also represented amongst the wealthiest and most distinguished, in the priesthood and in the royal household. It should be mentioned in passing that, at times, Negroid or Nubian princes occupied the Egyptian throne. This Negroid racial element was intimately intermingled with the second component of the Egyptian people. This is the Mediterranean race, the very same that inhabits south-western Europe. The Mediterranean race is likely to have constituted the bulk of the population, so that it is to be regarded as the main bearer of Egypt's Hamitic culture. But here too there were quite numerous admixtures of other races. A typical so-called 'Ramses nose' cannot be explained by either the Mediterranean or the Negroid race. On the other hand, Egyptian sources report centuries of fighting against the Semitic desert peoples who invaded from the east across the

Red Sea. In this way, the blood of the Oriental race entered the Egyptian people in large quantities. Furthermore, we learn that hordes of blond-haired invaders appeared by sea; thus, from time to time, Nordic influences may also have entered the population. Coloured illustrations depict individuals of the Nordic race. Finally, skeletal finds prove the existence of a race with a short skull; this is likely to be the 'Armenoid' type. — The small statuettes, which realistically depict dwarfish people, female figures with sagging breasts and the physical characteristic known as steatopygy (fat buttocks) found in modern-day Hottentot and Bushman women, show that the Egyptians must at least have been familiar with such people, making it likely that those African tribes once made their way up to this region. The racial composition of the Egyptian people is likely to have remained fundamentally the same from the very beginning to the present day, even if the numerical proportions of the individual mixed races have fluctuated, particularly as regards the Negro component. The further one travels up the Nile, the more the Negro race predominates; and to the south of Egypt lie regions where one must say that the inhabitants are either Negroes who have become heavily Mediterraneanised, or heavily 'Negroised' Mediterraneans.

From Egypt westwards, the Islamic population of Arab descent now forms the upper class throughout North Africa. But beneath them, particularly in Algiers and Tunis, the Mediterranean race—that is, the same people who inhabit southern Europe—is clearly recognisable as the so-called Berbers. Added to this mixture are, firstly, the Negro component, and secondly, the Oriental race brought by the Arabs. The proportion of these individual population elements varies considerably; at times one racial component predominates, at others the other. This population extends beyond the mainland to the Canary Islands off the coast of West Africa; the now-extinct Quanch people undoubtedly belong to this group, having perhaps preserved the ancient Mediterranean type and language particularly clearly. Hence certain affinities with the characteristics of the Cro-Magnon race. Southwards from the northern edge of Africa towards the desert, within the same mixture there is an ever-increasing predominance of Negro blood, a gradual transition to genuine Negro tribes, which, for their part, particularly in the distinguished families, repeatedly reveal traces of Mediterranean blood. Just as Hamitic and Semitic linguistic and cultural heritage can be recognised as far as the southern tip of Africa, grafted onto the Negro cultures, so too does Mediterranean and Oriental blood, mixed with Negro blood, extend that far.

The African Circle.

Anthropologically, the African circle encompasses Africa with the exception of the entire northern edge, that is, from the Sahara to the southern tip. Madagascar is included, whereas the West African islands are excluded. This region belongs to the race of the
'Negrides'. What ethnology refers to as 'Negroes'—that is, the entire cultural sphere of Negro cultures—presents an extraordinarily diverse picture from an anthropological perspective. In racial terms, as has just been shown, this population is strongly influenced from the outside by the Mediterranean and Oriental races. What has just been termed the indigenous 'Negroid' population consists, in turn, of two distinctly separate strata which have overlapped over time. The one, which may be described as more primitive, constitutes a Pygmy population. This includes individual groups living in tribes scattered across Africa: on the one hand, the Bushmen of South Africa; on the other, the so-called dwarf tribes in the western Congo and Cameroon forests, as well as numerous small tribes in the Central African jungle region and the East African lake region. These Pygmies are characterised by their short stature (average height for males: 1.40), round skull shape, convex upper lip and light skin colour

and were once widespread across the entire continent, even as far as Europe (figurines with steatopygy); today they have been driven back into desert and jungle regions.

The upper stratum, what is commonly called 'Negroes', varies extraordinarily from place to place in Africa, yet can still be recognised as various hybrid forms based on a single race. Anthropologically, there is no dichotomy of the sort that ethnology tends to apply to 'Negro' cultures—particularly in the linguistic sphere—by dividing them into Bantu and Sudanic groups. Tribes from the Sudanic group are, anthropologically speaking, mostly strongly 'orientalised' or 'mediterraneanised'; at other times, however, Bantu groups display exactly the same characteristics, and conversely, the true anthropological 'Negro' type is occasionally found in both Sudanic and Bantu groups in a distinct and pure form. This can be characterised as follows. Their height is very respectable, averaging between 1.62 and 1.82 metres for men across the various groups. Their proportions are characterised by the length of the arms and legs, particularly the forearms and lower legs. The skull is long and narrow, with a distinctive curvature of the forehead and occiput, a flat crown and a strongly prognathic face. The nose is exceptionally flat and broad, with nostrils that are almost horizontal; the cheekbones are prominent; the chin is distinctly defined; and the mouth is framed by thick, upturned lips. The skin colour is a dark brown in various shades; the hair and eyes are black. The hair is spiralled; body hair is relatively sparse; the beard appears late but is of European type. The mental faculties of the Negrids are distinctly different from those of Europeans, being peculiar and characteristically pronounced, though they cannot be described here.

These Negrids were once, just like the Pygmy population, spread across the northern edge of Africa and the southern edge of Europe (Grimaldi diffusion). Certain aspects of the culture of the ancient Central European Stone Age cultures of the Ice Age (Solutrean) also point to close ties between these Negrids and Europe. It was they who apparently subsequently displaced the Pygmies. Whether they, in turn, were pushed southwards by the Mediterranean peoples, or what the nature of the relationship between them and the Europeans was, remains unknown. The people most closely resembling them are found in the South Seas, where certain Papuan-Melanesians are of an extraordinarily similar type. Skin colour, hair type, nose shape and physiognomy resemble those of the Negroes extraordinarily closely in some respects, and to complete the resemblance, there are also Pygmies there, alongside the tall variety, who are referred to as Negritos. The relationship between them has not yet been clarified there either.

Finally, we should also consider the Negroes outside Africa, particularly those in the Americas. Through a mixture of all manner of African tribes, a distinctive intermediate type has emerged there, which has, however, retained the African characteristics—including the intellectual ones—well. Where these Africans have been left entirely to their own devices, as for example in Jamaica, individual groups have sunk into a state of complete cultural backwardness (fetishism, etc.) and have become so-called 'bush Negroes'. As is well known, in South America there has been an exceptionally high degree of intermingling between Europeans and Africans; there, the various degrees are distinguished as mulattos, tercerones, quarterones, etc.

East, Central and North Asian Region.

The bulk of the Asian continent (excluding India) is occupied by the 'Mongoloid' group. Anthropologically, the Eskimos are closely related to this group, whilst the indigenous peoples of the Americas are somewhat more distantly related

indigenous peoples. Ethnology classifies them into Mongols in the narrower sense, comprising a northern branch—the Manchus and Koreans—and a southern branch—the southern Chinese and Japanese—followed by Turkic-Tatar and, finally, Ural-Altaic tribes. An anthropological classification of this vast mass of people has not yet been possible, but the aforementioned ethnic groups are also distinguished from one another by a multitude of anthropological characteristics, so that the Mongoloids as a whole certainly break down into a series of sub-races, without those boundaries corresponding entirely to the former. Common to all is the Mongolian face: a flat countenance with prominent cheekbones, the flatness of which is due to the fact that the zygomatic bones run parallel and straight forwards before bending almost at right angles. The bridge of the nose is low; the nose itself is broad and flat, though by no means as broad as the Negroid nose. The eye has the so-called Mongolian fold described above (page 91). The skin is characterised by a distinctive yellow-brown pigment, ranging from a lighter yellow in some groups to a deep brownish-yellow in others. The Mongolian spot (see p. 92) is characteristic of all of them. The hair is coarse; the individual hairs are very thick, hard and, in cross-section, almost square with rounded corners. Body height varies. Among northern Chinese groups, the average height for men is 1.70 m; among the Tartars, 1.63 m; among the shorter Japanese, 1.58 m; and among the Lapps, occasionally 1.52 m. The shape of the head also varies somewhat. It is generally of medium width to broad. Among the Japanese, it has been possible to demonstrate very clearly – just as among us Europeans – a more refined type among the socially upper classes and a coarser type among the lower classes. Among the former, the faces become narrower and longer, the nose occasionally small and, by Japanese standards, downright narrow.

The Mongoloid race possesses an immense capacity for expansion. From the Asian continent, it spread in prehistoric times – perhaps in connection with the Aryan migration to India – via the so-called Malay Archipelago, where it underwent an extraordinarily large number of intermixings of all kinds and degrees with the indigenous population, which was itself very diverse. This expansion took place in two waves: an earlier one, perhaps also smaller in number, merged much more closely with the indigenous population and gave rise to what are today known as the ‘inland’ or ‘original’ Malays, who in some places display no, or only very few, Mongoloid features. The second wave gave rise to the so-called Deutero- or coastal Malays—those we now call Javanese, Tengger, etc.—who, for the most part, still display Mongoloid features quite clearly, mixed with those of the indigenous population.

Another powerful wave, perhaps earlier than the first, moved westwards and brought about the Mongolisation of the Slavs, with all the consequences described above. And a most recent wave is heading eastwards to the west coast of America – the 'Yellow Peril' facing America today! The settlement of the Japanese islands, too, took place only as part of a secondary migration from the mainland; the ancient indigenous population was partly absorbed into the Japanese, particularly the southern group, whilst the northern group survived on Sakhalin and Yesso as the small Ainu people. The Ainu show no trace of Mongoloid characteristics; rather, with their thick beards and straight-set eyes, they resemble Europeans more closely. They may well have sprung from the same roots as the European race and been driven across Asia to their present-day habitats. Certain Indian groups, such as the Toda, may have originated in a similar manner.

The Eskimos have a special relationship with the Mongoloids; they differ from them in certain distinctive cranial features, particularly a large degree of brachycephaly, but are Mongoloid in all other respects. They may have separated from the Mongoloid stock very early on and then developed separately in their Arctic isolation.

Likewise, the indigenous peoples of the Americas are thought to have originated from the Mongoloid stock and, as linguistic and ethnographic research demonstrates, to have migrated to the Americas via the Bering Strait after the Ice Age. There, they spread across the entire continent, forming distinct racial subgroups in the various regions. They have retained their yellow-brown pigmentation, Mongoloid proportions and the Mongolian spot; as for the Mongoloid epicanthal fold, they either did not possess it at that time or have lost it entirely. A particularly striking distinctive feature is the narrowness and length of the nose, which is characteristic of all Native Americans and has, in some individuals, resulted in particularly pronounced forms of the aquiline nose.

The Australian-Pacific region.

To conclude, we shall cast just a very brief glance at the Pacific islands and Australia, merely to highlight the adaptability of the human species and the diversity of racial formations. On the Indian mainland, in Ceylon and on certain Malay islands live the remnants of the Wedda racial group, a particularly primitive form— ly small, though not quite pygmy in stature, with broad faces, low noses and wavy hair similar to that of Europeans. Parallel to them, not dissimilar in type but nevertheless clearly distinct due to skull shape, considerable height and a different beard pattern, is the Australian race, which, judging by skull structure, has remained the most primitive among those existing today. Alongside the Wedda group, the aforementioned Negrito group—distinct from the former by their strongly frizzy hair—are the Papuan Melanesians, and, probably constituting a separate group, the Tasmanians, who also have frizzy hair but differ from the Negritos in body size and skull shape.

Finally, standing in contrast to these, with straight hair and lighter skin, unlike the rest of the South Sea population, are the Polynesians, who in many respects—body proportions, physiognomy and hair type—resemble Europeans. All these are, it seems, branches that sprang independently from the root of humanity and have, in some cases, remained in a primitive state, whilst in others they have developed in a distinctive manner. Countless interminglings took place between these South Sea tribes.

Section Three.

Pathological hereditary traits.

By **Dr Fritz**

Lenz, private lecturer at the **.**

1. On the concept of disease.

It is characteristic of living beings that they are generally adapted to their usual environment, both in their structure and in their modes of reaction; and we call a living being adapted to
its environment, provided that its structure and the associated manifestations of life within that environment ensure the preservation of life.

It is not uncommon for us to encounter living beings which, whether as a result of external influences or the nature of the organism itself, lack this adaptation to a greater or lesser extent, and in which the preservation of life is therefore compromised. We describe the condition of a living being that lives at the limits of its capacity for adaptation as 'sick'. There are therefore all degrees of transition between full health and the most severe illness. There is no distinction between health and illness in a biological organism.

> For a long time, it was thought that every species of living being had a specific 'normal range of variation' and that deviations from the average type within a certain margin were to be regarded as normal, whilst deviations exceeding this 'normal range of variation' were to be regarded as pathological. However, no such boundary can exist; for when asked about the limits of the 'normal range of variation', one cannot answer that it ceases where the pathological begins. Nevertheless, it is important to clarify in what respect the pathological differs from the healthy. As a conceptual yardstick for this, we take the probability of survival, and the resulting definition of disease is consistent with both common parlance and that of medical science.

Deviations from the population average are considered pathological if they result in a below-average probability of survival for those affected. Not all deviations from the mean are pathological. Even a particularly strong person deviates from the mean in terms of physical strength. In general, however, the degree of pathological nature increases with the deviation from the mean. Let corpulence serve as an example. Most people have an average level of corpulence. High degrees of emaciation, however, are a sign of pathological nature, and

likewise, high levels of obesity are pathological and life-threatening. We therefore understand 'disease' to mean the condition of an organism in the limits of its capacity to adapt. Full health denotes the state of complete adaptation, and the more a living being's adaptation is impaired, the more seriously ill it is. If it is pushed beyond the limits of its capacity to adapt by internal or external causes, death ensues. The dead body no longer exhibits any adaptive responses; this distinguishes it from the living body.

As a rule, the causes of a disease are not to be found exclusively in either environmental influences or genetic factors; rather, both groups of causes usually interact. It is, however, relatively rare for both groups to be of roughly equal significance; as a rule, one or the other tends to be practically decisive. Thus, in the case of most so-called infectious diseases, the decisive cause lies in the invasion of certain microorganisms into the body and their multiplication at the body's expense. By 'hereditary diseases', on the other hand, we mean those in which pathological genetic factors play the decisive role in their development.

> It has been argued that hereditary diseases cannot possibly exist, because a disease is always a process and never a state, and because processes cannot be hereditary. This objection seems to me to stem from an inappropriate definition of the concept of 'disease' by the pathologist Virchow, who rightly enjoyed great prestige in the 19th century. On closer inspection, the question of the 'nature of disease' is merely a question of definition or terminology. Definitions are in and of themselves neither 'true' nor 'false', but merely more or less appropriate for their intended use. If one were to declare it inadmissible to speak of 'hereditary diseases', one would have to strip diabetes and many other conditions—which are designated as 'diseases' not only in everyday life but also in medical science—of this name. We consider it more appropriate to define the concept of disease in such a way that it also encompasses these conditions. Pronounced pathological processes, of which pneumonia usually serves as a model, are likewise encompassed by our concept of disease. For when a person is stricken with pneumonia, they belong, in terms of their current condition, to the weak variants—specifically, the paravariants—and accordingly, the threat to their life is great.

Pathological hereditary predispositions follow exactly the same laws of inheritance as normal ones. For anyone who has realised that there is no biological

fundamental difference, this is nothing more than self-evident. Because certain pathological traits distinguish their carriers markedly from the rest of the population, it has been possible to trace their inheritance particularly well. This is why it is precisely through pathological traits that the validity of Mendel's laws for humans has been best demonstrated. It is also related to this that the inheritance of the vast majority of hereditary conditions is relatively straightforward. Pathological genetic traits are usually either simply dominant, simply recessive or sex-linked recessive. It would, of course, also be conceivable that certain genetic traits, which on their own do not result in any, or at least no serious, pathological condition in the carrier, only lead to serious illness when they interact with others. This may be true in some cases; it is certainly not the rule, and no such instance has been proven to date. Consequently, although complex segregation patterns, as described in the general section, are of great significance for the heritability of differences in intellectual ability—which will be discussed later—they are not relevant to hereditary diseases. In the case of the latter, the circumstances are in fact considerably simpler.

2. The significance of pathological hereditary factors for diseases of the various organs.

a) H e r e d i t a r y eye disorders.

A discussion of hereditary diseases and disorders is best begun with those affecting the eye. No other organ is as well understood in terms of pathological hereditary factors as the organ of sight, and this is no coincidence. The eye is the most complex and important of our sensory organs; even relatively minor anatomical deviations in its structure can result in considerable impairment of function. Furthermore, the eye, unlike any other organ, is clearly visible and accessible to medical examination. Finally, there appear to be a particularly large number of ophthalmologists who have a penchant for rigorous scientific work, and as their field of specialisation is not very broad, many have devoted themselves in an exemplary manner to researching the hereditary causes of eye disorders.

It has long been known that so-called refractive errors of the eye are, to a large extent, hereditary. The refractive state of the eye depends on the interaction of several parts of the eye: the length of the eyeball, the curvature of the cornea, the curvature of the lens, and so on. When the eye is to be focused on nearby objects, the lens must exert a curving force; otherwise, the image of nearby objects would fall behind the light-sensitive retina, as follows from the laws of refraction. The eyes of a not inconsiderable proportion of people are, however, already focused on near objects even at rest. This condition is referred to as short-sightedness or myopia. Consequently, these eyes are unable to focus clearly on distant objects. There are varying degrees of short-sightedness; the milder forms do not cause significant visual impairment, particularly as vision can be easily corrected with suitable spectacles

. Severe short-sightedness, which tends to result from an increasing elongation of the axial length of the eye, on the other hand, constitutes a serious pathological disorder that can even lead to blindness.

Until recently, the view that short-sightedness was caused by strenuous and prolonged close-up work was fairly widespread. As a marked increase in short-sightedness is often observed during school age in particular, the term 'school myopia' was even used. This theory has now been overturned, particularly by very extensive research and astute analyses by the Zurich ophthalmologist Steiger. It may be regarded as established that the increase in short-sightedness during adolescence is due to internal causes. The statistical evidence that has been put forward to support the occurrence of myopia acquired through school or occupational work is, in its entirety, unsound. The notion that short-sightedness is, as it were, a fixed adaptation to close-up work must be abandoned without reservation. Above all, it is entirely mistaken to attribute the initial development of the hereditary predisposition to short-sightedness to such a supposedly individually acquired adaptation. Such notions are entirely incompatible with the findings of hereditary research (see pp. 16–21)

Most ophthalmologists and school health specialists today seem to adopt a middle-of-the-road position, assuming that whilst the predisposition to short-sightedness is hereditary, this predisposition can then be brought to fruition by close-up work. Although this obvious and seemingly cautious assumption is difficult to refute, it can by no means be regarded as established. There is no evidence whatsoever that myopia can even be exacerbated by close-up work. As for its hereditary nature, however, there can be no justified doubt. A large number of family trees have been published, one of which I reproduce here in abridged form, following L u t z:

Fig. 14.
Family tree of a family with short-sighted members. After Lutz (abridged). = short-sighted in one eye.

The predisposition to short-sightedness found in this family is apparently simply dominant; that is to say, it manifests itself in every individual who carries it; every short-sighted family member has at least one short-sighted parent; conversely, if both parents have normal vision, then none of the children will be short-sighted.

It is likely that different families also have different pathological predispositions that cause short-sightedness. Many of these predispositions appear to be dominant; however, it is by no means necessary that they all are. The predisposition to severe, progressive short-sightedness, in particular, does not appear to be dominant. Whilst it is certain that this too is idiotypically determined, we do not yet know the pattern of inheritance.

Just as with short-sightedness, long-sightedness or hyperopia has also been shown to be hereditary, i.e. the condition in which the image of distant objects also falls behind the retina. In order to see clearly, farsighted people must therefore exert the lens to change its curvature even when looking into the distance, and naturally even more so when looking at close objects, which, in more severe cases, is associated with considerable discomfort. Incidentally, when the eye is at rest, the image of distant objects seems to fall behind the retina in most people. However, as even a very slight curvature of the lens is sufficient to bring about the correct focus, mild hyperopia—unlike mild myopia—is not to be regarded as a pathological condition.

Furthermore, astigmatism is also hereditary; this is a condition in which the cornea is more curved in one direction than in another, and as a result, all objects are seen out of focus. Since, according to Steiger, the degree and type of curvature of the cornea are largely hereditary, it follows that astigmatism is also hereditary, just like other refractive errors.

> Of course, one must not assume that only pathological deviations from the normal state of refraction are hereditary; rather, this normal state itself is, of course, also hereditary, namely through the interaction of a large number of genetic units. If any one of these genetic units undergoes an idiovariation, a hereditary refractive anomaly arises. Milder anomalies can also arise when incompatible genetic units occur together in an individual. Thus, a particular corneal curvature may result in normal refraction for a certain axial length of the eye, but cause short-sightedness for a greater axial length; and the axial length that contributes to short-sightedness in this case may, when combined with a different corneal curvature, result in normal vision once again. The refractive state of the eye is therefore also influenced by mixed variations which, whilst not being passed on as such in the inheritance process, are nevertheless idiotypically determined.
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> The inheritance of eye colour has already been discussed above (p. 98). In the realm of pathological conditions, however, heterochromia falls, in which one eye is dark and the other blue. The light-coloured eye in such individuals is prone to a peculiar, insidious inflammation which can lead to the loss of sight. In cats, where heterochromia has been selectively bred, deafness is said to occur regularly on the side of the

blue eye. Cats with blue eyes in both eyes are generally deaf. In humans, too, heterochromia has been traced over several generations. It appears to be a dominant genetic trait which inhibits the development of pigment in one eye. In any case, it is not the case that heterochromia could arise from the cross-breeding of individuals with light-coloured eyes and those with dark-coloured eyes; rather, in all such cross-breedings, both eyes always retain the same colour.

Fig. 15.
Family tree of a family with a l b i n o members, according to T e r t s c h.

If the pigment of the eye is wholly or almost wholly absent, this is referred to as o c u l a r a l b i n i s m. This condition occurs as part of g e n e r a l a l b i n i s m, in which the hair is snow-white to yellowish-white from childhood onwards due to a lack of pigment. The pigment-free iris then appears reddish due to the blood from the blood vessels showing through, as does the normally black pupil, because the retina is also devoid of pigment in this case. As the protective effect of the dark pigment is absent, such individuals suffer from painful sensitivity to bright light. Such 'cockroaches' therefore usually keep their heads lowered and their eyelids almost closed. This sensitivity to light is often accompanied by rhythmic twitching of the eyeball (nystagmus). It is precisely this susceptibility of the eyes to being dazzled by light that underlies the pathological nature of albinism, which justifies its discussion here. As a partial manifestation of general albinism, the ocular form is also simply recessive, as the accompanying family tree illustrates; this tree is also instructive regarding the inheritance of recessive traits in general.

In this family tree, on the left, we see a marriage between an uncle and his niece, from which four albino and two normal children are born. The albino mother is to be regarded as homozygous, the father as heterozygous. Through this marriage between relatives, the recessive trait for albinism has been brought together from both sides. In the centre of the family tree, we see one albino child and two normal children resulting from a marriage between first cousins. As both parents are healthy, they are to be regarded as heterozygous. The fact that the trait cannot be dominant follows from the fact that, in three marriages, albino children are born to parents who are healthy on both sides. General albinism is, rather, to be regarded as recessive and appears to be due to the absence of a genetic unit, which was designated 'A' earlier on p. 36 in the case of rabbits. The pedigree illustrates very clearly the significance of consanguineous marriage in the manifestation of recessive conditions. It is reported that approximately one third of all individuals with albinism are born to consanguineous parents, whilst the frequency of consanguineous marriage otherwise accounts for only a few per cent.

Apart from general albinism, which also affects the eyes, there is also a form of albinism confined to the eyes. Skin and hair colour are completely normal in this case; the pathological

manifestations in the eyes are, however, the same as those just described. It is very curious, however, that those affected by this condition are invariably men. No cases of women with isolated ocular albinism have been reported to date. Healthy women, however, can pass on the predisposition to their sons via their father; indeed, as theory suggests, on average to half of their sons; daughters are, of course, spared; yet the condition can be passed on latently through the female line across two, three or even more generations, until it eventually manifests itself in male descendants. Men can never pass the condition on to their sons , but they can do so indirectly via their daughters to male grandsons. We refer to this mode of inheritance as sex-linked recessive. As with ordinary recessive inheritance, the parents of affected individuals are generally normal. However, there is also the peculiar link to sex, which can be explained quite simply if one assumes that the pathological predisposition is due to the absence of a genetic unit that is normally present in the sex chromosome (cf. p. 57). Since the male has only one sex chromosome, such an effect manifests itself immediately; in females, on the other hand, who have two sex chromosomes, the defect in one sex chromosome is masked by the corresponding normal genetic unit in the other sex chromosome. The following family tree serves to illustrate this pattern of inheritance:

Fig. 16.
Family tree of a family in which several members suffer from limited albinism with nystagmus. According to Mansfield (abridged; the descendants of some second-generation women have been omitted).

In this family tree, we can see how the grandfather's condition is present in several male grandsons, but is also passed on to a great-grandson via two generations of the female line. The fact that the condition is not dominant is evidenced by the fact that the pattern of inheritance cannot be traced in an unbroken line, as is the rule with dominant conditions.

The same mode of inheritance as ocular albinism applies to so-called megalocorne, an anomaly in which the cornea is unusually large and the entire eye therefore appears very large.

A blue-grey colour of the sclera of the 'white' of the eye occurs as a secondary manifestation of an apparently dominant constitutional anomaly, the pathological significance of which lies in abnormal fragility of the bones (see below).

A large number of minor and major eye malformations are distinctly hereditary. Relatively harmless is what is known as d i s t i c h i a s i s, a condition in which a second row of hairs grows along the lower edge of the upper eyelid, behind the eyelashes, in place of the glands that would normally be found there. This anomaly is dominant. The same appears to apply to what is known as epicanthus, a wrinkled fold between the upper and lower eyelids that extends across the inner corner of the eye (see p. 90). This anomaly is often accompanied by a condition in which the upper eyelid droops and cannot be raised. This condition, known as ptosis, also occurs as an independent dominant anomaly. Similarly, a whole range of other defects affecting the eye muscles and their nerves are hereditary. A significant proportion of all cases of so-called strabismus are attributable to such defects. Another type of strabismus is associated with hyperopia. Focusing on near objects is normally accompanied by an inward movement of the eyes. Since the effort required to focus is particularly great in cases of severe hyperopia, it is understandable that the eye axes may be turned too far inwards in the process. This is the most common form of strabismus, and it is hereditary, occurring alongside severe hyperopia. The deviation of one or both eyes outwards also occurs as a hereditary condition. In cases of unilateral amblyopia, the affected eye frequently deviates from the line of sight; thus, hereditary amblyopia in one eye can also lead to hereditary strabismus. However varied the forms of strabismus may be, it can nevertheless be said that, in its aetiology, external (parakinetics) causes take a back seat to hereditary (idiotypic) ones.

A condition also occurs on a hereditary basis in which all the external eye muscles are paralysed, so that the eyes cannot be moved (external ophthalmoplegia). The condition has been observed in an unbroken line through five generations and is therefore evidently simply dominant.

Several forms of peculiar corneal opacities are idiotypically determined; Fleischer summarises these as ‘familial corneal degeneration’. At birth, the eyes are still clear; however, as the child grows, opacities develop which gradually become denser until, eventually, vision is almost completely lost. The pattern of opacity varies between different families (being either nodular, latticed or patchy), but remains consistent within the same family. It appears to involve several dominant anomalies.

Complete absence of the iris (aniridia or irideremia) is inherited in a dominant manner. Much more common is partial absence of the iris, in particular downward slits known as colobomas, which are also inherited dominantly.

On an idiopathic basis, a congenital condition also occurs in which the lens is not in its correct position, which naturally results in severe visual impairment. Curiously, such a congenital displacement of the lens was found in one family to be associated with a predisposition to severe heart disease (cf. p. 69).

Abnormal smallness of the eye (microphthalmia) has been observed once across three generations, and on several other occasions among siblings.

Even the complete absence of the eyes (anophthalmus) appears to occur as a hereditary condition, as it has been observed on several occasions in newborn children within the same family. The fact that such individuals do not reproduce does not refute the hereditary nature of the predisposition. Dominant inheritance is, of course, out of the question, but the assumption of recessive inheritance seems entirely possible. It is not even impossible that such an extreme malformation as cyclopia—in which the two eye buds fuse in the centre to form an organ that is naturally non-functional—might be hereditary. Until now, in the case of such severe malformations in non-viable foetuses of healthy parents, the possibility of their being hereditary has usually not even been considered, precisely because one has proceeded from a far too narrow concept of heredity. When one hears the word 'heredity', one all too easily thinks of the transmission of characteristics from ancestors to descendants. If one wishes, in a strictly biological sense, to describe the transmission of idioplasmic predispositions inherited from ancestors (cf. p. 5), one would do well to use the term 'idiophory', as proposed by Siemens.

Fig. 17.
Family tree showing recessive retinal atrophy.
After Boehm.

On a hereditary basis, blindness may also develop over the course of a lifetime in individuals born with apparently normal eyes.

This can occur in a variety of ways. Blindness associated with so-called amaurotic idiocy and related hereditary conditions will be mentioned only briefly here, as these conditions are discussed under mental disorders (see p. 227).

It is not entirely uncommon for blindness to result from hereditary retinal atrophy ('retinitis pigmentosa'). The condition begins in early youth with a narrowing of the visual field, which progresses steadily over the years until, after decades, vision is finally lost even in the centre of the visual field. There are several types of this clinical picture, grouped according to their clinical manifestations, which do not appear to differ significantly in terms of outward appearance or course, but which are nevertheless idiotypically

, as is evident from their different modes of inheritance. A recessive form is relatively the most common.

It is linked to the recessive mode of inheritance that those suffering from retinal atrophy are strikingly often the product of consanguineous marriages; according to some reports, this accounts for more than a quarter of cases. In the family tree shown, we can see how, from the marriages of two brothers to two sisters—who are their first cousins—one person suffering from retinal atrophy emerges from each union.

A sex-linked recessive form of retinal atrophy is less common, as shown in the following family tree:

Fig. 18.
Family tree showing X-linked recessive retinal atrophy. According to Nettleship (excerpt).

Finally, there is also a simple dominant form:

Fig. 19.
Family tree showing dominant retinal atrophy. Adapted from Nettleship (excerpt).

Hereditary diseases originating in the choroid (Chorioidea) are rarer than hereditary retinal diseases. For instance, a dominantly inherited total choroidal atrophy has been described. As the overlying retina is also affected in this condition, the disorder may be identical to dominant retinal atrophy.

The retina can be regarded as an extension of the optic nerve (nervus opticus); and like the optic nerve, it is also frequently the site of hereditary disorders. For instance, defects in the optic nerve (optic colobomas) have been described as arising from a hereditary predisposition. The pattern of inheritance of idiopathic optic nerve atrophy ('optic neuritis') is relatively well understood. The condition usually first manifests in the second to fourth decade of life. In cases of inflammatory involvement of the optic nerve, both eyes may lose their vision within a few days, specifically in the very centre of the visual field, where vision is normally sharpest. More commonly, however, the progression is slower. A residual degree of vision is regularly preserved in the peripheral parts of the visual field, which is precisely the opposite of what occurs in pigmentary retinopathy. Within the same family, the course of optic atrophy tends to be fairly consistent. In most families, only men are affected. The mode of inheritance is X-linked recessive.

Fig. 20.
Family tree of a family with optic nerve atrophy. After Hensen.

It seems doubtful whether, in individual families, the genetic predisposition to optic nerve atrophy can also lead to the disease in female individuals who are heterozygous for the condition. It is difficult to determine this with certainty, as optic nerve atrophy can also be caused by external factors (infections, poisoning).

A rare form of optic atrophy occurs in association with a peculiar, apparently dominant malformation of the skull, known as 'tower skull'. In this condition, the frontal region is protruding forwards and upwards, and it appears that the optic nerve is then destroyed by pressure resulting from bone growth during the first years of life.

A relatively common cause of blindness in middle age is so-called glaucoma, which appears to be due to a pathological increase in pressure that causes the retina to atrophy. A distinction is made between 'acute' or 'inflammatory' glaucoma, in which vision can be lost within a few days amidst severe pain, and 'chronic' or 'simple' glaucoma, in which vision is lost over a longer period amidst less severe or only occasional pain. The course of the disease is generally quite similar among members of the same family.

Fig. 21.
Family tree of a family with inflammatory glaucoma. After How.

When examining family trees of glaucoma, one almost always finds a pattern of inheritance in an unbroken line. It therefore appears that anyone with a predisposition to the condition will, sooner or later, develop the disease. Glaucoma, particularly the inflammatory form, is most commonly found in psychopathic individuals. There are families in which it regularly occurs alongside constitutional imbalance and anxiety about the heart, an example of how various physical and mental disorders can be dependent on a single genetic factor.

Hydrophthalmus ('watery eye'), which is also hereditary, is in some ways similar to glaucoma; it is caused by the absence of the so-called Schlemm's canal, through which the aqueous humour normally drains. As a result of this defect, fluid accumulates in the eye, causing it to swell to such an extent that vision is lost as early as childhood.

It has been claimed of glaucoma, as of some other hereditary conditions, that the disease occurs at an increasingly earlier age in later generations than it did in the ancestors from whom the predisposition originates. Scientific-sounding terms have also been coined for this, namely 'anticipation' or 'anteposition'. When examining family trees of glaucoma sufferers, one can indeed usually observe

that the ancestors did not develop the condition until they were 40 or 50 years old, or even later, whereas living family members develop it as early as 30 or even 20 years of age. The findings of modern genetic research, however, clearly contradict the assumption that this is a biologically determined phenomenon; it is more likely to be a statistical illusion. As blind people very rarely marry, it is generally only those individuals with a predisposition to glaucoma who enter into marriage, in whom the disease does not manifest until later in life. The affected ancestors thus represent a selection based on late onset of the disease, whilst in the living generation, conversely, it is precisely those individuals in whom the condition manifested at an early age who are found to be affected, whereas those siblings who do not develop the condition until later are still found to be healthy. This gives rise to the impression of 'anticipation', without there necessarily being any biological basis for it.

'Anticipation' has also been claimed for those forms of hereditary cataracts that only develop over the course of a person's life. Any clouding of the lens, whatever its cause, is, as is well known, referred to as a cataract. The common so-called age-related cataract, which usually only occurs in old age, was until very recently often regarded exclusively as a consequence of ageing. However, there are quite a few people aged 80 and over who show no sign of a cataract, whilst in others it occurs as early as middle age. At the same time, external influences such as intense heat in the case of fire-workers can promote the formation of cataracts; yet the same harmful effects may act much more rapidly and destructively in one person than in another. On the other hand, there are entire families affected by cataracts where lens opacity occurs as early as the second or third decade of life, without any evidence of external harmful factors. It is therefore likely that, at least in the case of age-related cataracts, susceptibility is hereditary.

Heredity is particularly pronounced in congenital forms of cataract, of which a whole range have been distinguished. In most cases, the lens is not completely but only partially clouded, e.g. only the innermost nucleus ('central cataract') or only a zone around the nucleus ('zonular cataract'). Congenital forms of cataract are largely dominant.

Fig. 22.
Family tree of a family with congenital central cataract. After Nettleship (detail).

Within the same family, the age at which the condition manifests remains fairly consistent. It is not the case that the age-related cataract of earlier generations progresses to juvenile cataract and eventually to congenital cataract in subsequent generations. Nor, of course, does it make any difference to the children's prognosis whether they are born before or after the development of the predisposition to cataracts in their parents.

Congenital cataract is one of the principal causes of congenital blindness. Of those who are blind in childhood, about a quarter were born blind; and congenital blindness is almost always idiopathic in nature. In the case of acquired blindness, on the other hand, external causes predominate (gonorrhoeal infection of the eyes at birth, injuries, etc.). Overall, there were around 30,000 blind people in Germany before the war, of whom several thousand at least owed their disability to pathological hereditary factors.

Cataracts are often merely a secondary symptom or a consequence of general conditions associated with metabolic disorders. It occurs quite frequently in hereditary diabetes (see p. 203), and also in chronic tetany, a severe neurological disorder accompanied by convulsions; in the myotonic dystrophy described by Fleischer (see p. 222), it occurs regularly.

The link between eye disorders and hereditary systemic conditions is, in general, very strong ( ). Worth mentioning is the protrusion of the eyes in Graves' disease ('exophthalmia', see p. 195). In hereditary migraine, a so-called scintillating scotoma is often found, which usually presents as a spot dancing back and forth in the visual field and which also appears to occur hereditarily even in the absence of migraine. The connection between hereditary eye disorders and general disorders reflects the fact that, strictly speaking, every genetic unit affects the entirety of an organism's genetic makeup; or, which amounts to the same thing, that the structure of every organ is determined by the entirety of the genetic makeup, although the effect of a genetic unit tends to manifest itself predominantly in individual organs.

Pathological genetic predispositions also play a significant role in the development of inflammatory eye conditions. This appears to apply, for example, to chronic inflammation of the eyelid margins (blepharitis ciliaris) and to chronic inflammation of the lacrimal sac (dacryocystitis), both of which occur with increased frequency within families. Susceptibility to so-called 'spring catarrh'—which is based on a pathological sensitivity of the conjunctiva to sunlight—and to hay fever—which is based on a pathological sensitivity to grass pollen—is also likely to be at least partly idiotypically determined.

Also worthy of mention is the hereditary nature of a malignant tumour of the retina, the retinal glioma, which develops in early childhood and, following the destruction of the affected eye, ultimately leads to death. In one family, all eight children were affected by it; in another, all four; and in a third, four out of seven.

In a number of hereditary disorders of the visual organ, no pathological structure can be detected at all using our current diagnostic tools; rather, the pathology manifests itself in these cases solely as certain disturbances of the sense of sight, particularly in the loss of certain functions.

This group includes what is known as night blindness or hemeralopia, of which two idiotypically distinct types are known. Common to both is that those affected are unable to find their way in conditions of greater twilight, whilst during the day they see just as well as others. Thanks to the English ophthalmologist Nettleship, a very extensive family tree relating to night blindness has come to light, spanning ten generations and comprising 2,116 individuals, 135 of whom are night-blind. It is the largest family tree known to date concerning a hereditary condition. For each of these night-blind individuals, the pathological predisposition can be traced back in an unbroken line to the night-blind butcher Nougaret, born in 1637 in Vendemian near Montpellier. Every night-blind member of this family circle therefore has at least one night-blind parent; in one case, both parents were night-blind and had two night-blind daughters. Where neither parent was night-blind, the children were, without exception, free from the condition. The predisposition is therefore dominant. To illustrate this, I present two extracts from the Nougaret family tree.

Figs. 23 and 24.
Families with d o m i n a n t n i g h t b l i n d n e s s. (Excerpts from the Nougaret family tree according to N e t t l e s h i p.)

In one part of the family tree reproduced here, we see the majority of members affected by the condition; in the second, only a minority. However, this does not reflect any biologically determined difference. According to Mendel's laws, one would expect that, on the whole, half of the children would be night-blind and half would be normal. A single child therefore has a probability of ½ of becoming night-blind, and each of its siblings has the same probability, independently of this. Consequently, according to the laws of probability, in such an extensive family circle, one would expect to find branches with many affected members alongside those with few. The two sections of the family tree have been deliberately selected to show that, amongst the offspring of a single pair of parents, the Mendelian numerical ratios need not hold true, even though the distribution of the trait in question as a whole does indeed follow Mendel's laws.

Apart from this dominant form of night blindness, there is another form which, unlike the former, is regularly associated with short-sightedness.

Several family trees are known for this form, which show that it is sex-linked recessive.

Fig. 25.
Pedigree of a family in which night blindness associated with short-sightedness is inherited in a sex-linked recessive manner. According to Cutler.

This pedigree can also serve to illustrate that there is also a sex-linked recessive predisposition to short-sightedness.

One can imagine that the type of night blindness just discussed is due to the absence of a gene on the sex chromosome (cf. p. 152), whereas dominant night blindness is due to the presence of an abnormal gene on some other chromosome, which inhibits the development of the ability to see in twilight. Vision in deeper twilight relies on different retinal elements than vision during the day. Daytime vision is facilitated by the so-called cones of the retina, which also mediate colour perception. Vision in deeper twilight, on the other hand, is facilitated by the so-called rods of the retina, which convey pale, colourless images, such as those we recognise from moonlit landscapes. The colour-sensitive cones require a greater intensity of light to function; they are therefore inactive in the dark.

There is, however, a hereditary condition in which, in the
In contrast to night blindness, it is specifically the function of the fovea that has failed. The individuals concerned therefore retain only that part of their sense of sight which enables us, for example, to find our way in the moonlight, but which, in bright daylight, is obscured by glare
is rendered inoperative. This condition is therefore referred to in England as 'day-blindness' or, in our country (less accurately), 'total colour-blindness', because the loss of conal function also results in the loss of all colour perception. The condition is recessive:

Fig. 26.
Family tree of a family with day blindness ("total colour blindness"). According to Hessberg.

We can see from this family tree how a marriage between first cousins—which is, of course, of great significance for the development of a homozygous recessive trait—results in three children with colour blindness. Naturally, it is a relatively rare coincidence that, out of four children here, three are affected, because with recessive conditions, the probability of a child developing the condition when both parents are heterozygous is only
¼. We should therefore not be surprised that, in some families with a larger number of siblings, only one is affected by a recessive hereditary disorder, as the following family tree illustrates:

Without any other experience of colour blindness and without a certain knowledge of the theory of inheritance, one would hardly think that such an isolated case within a family could be hereditary. Even today, in cases such as this – where the parents, grandparents and all eight of the sufferer's siblings are healthy – some doctors still tend to conclude that 'heredity' is not a factor.

Fig. 27.
Family tree of a family with a colour-blind ('totally colour-blind') member. According to Lutz.

The colour-sensing cones of the eye, which are completely non-functional in total colour blindness, lose their function only partially in other genetic disorders. The perception of blue is mediated by different elements to the perception of green or red. The loss of individual elements results in partial colour blindness. Red-green colour blindness (dichromacy) is particularly common, affecting around 4 per cent of all men and 0.4 per cent of all women. It is inherited in an X-linked recessive pattern.

There are two different forms of red-green colour blindness, one of which is sometimes (albeit somewhat inaccurately) referred to as 'red colour blindness' (protanopia), and the other as 'green colour blindness' (deuteranopia). As red and green are antagonistic colours that are mutually dependent in perception, if the perception of red is lost, green cannot be perceived as a colour either, and vice versa; however, when exposed to long-wavelength (red) light, a person with green-colour blindness experiences a stronger (colourless) sensation of brightness than a person with red-colour blindness. A person with red-colour blindness sees colours in much the same way as a person with normal colour vision does when it is not too dark. As twilight deepens, a person with normal colour vision also loses the colour perception associated with the cone cells, starting with red, whilst blue and yellow are initially retained. As twilight deepens, all colour perception is then lost even for people with normal vision, until, in moonlight, they see just as those who are completely colour-blind do. There are also abnormal genetic conditions which do not result in a complete loss of red and green perception, but only in a weaker development of the relevant functions. This is referred to as colour weakness or anomalous trichromacy. Nothing definite is known about its mode of inheritance, but it is presumably the same as that for red-green colour blindness.

Fig. 28.
Family tree of a family with red-green colour blindness. According to Groenouw.

Whilst in most other cases of X-linked recessive inheritance, affected females are not known to exist, women with red-green colour blindness are not entirely uncommon. Nagel has published such a family tree.

According to the theory of sex-linked inheritance,

Fig. 29.
Family tree showing the presence of red-green colour-blind women. According to Nagel.

we must assume that, in this family, a red-green colour-blind man married a woman with a latent predisposition to the same condition. In that case, each of the female children has a probability of ½ of being red-green colour-blind

. The sons of a red-green colour-blind woman must all likewise become red-green colour-blind, and the family tree is consistent with this as well. If a red-green colour-blind man were to marry a woman with the same condition, all their children would also have to be red-green colour-blind. In humans, such cases—which are, of course, extremely rare—have not yet been found. In animal breeding experiments, however, it has been possible to demonstrate all the possibilities of sex-linked inheritance (see p. 55 ff.).

According to the theory of sex-linked inheritance , the probable frequency of red-green colour blindness in females can be calculated from its frequency in males. Since approximately 4 per cent of all men are red-green colour blind, roughly one in every 25 sex chromosomes carries a corresponding defect. Such a sex chromosome therefore has a probability of 1/25 × 1/25 = 1/625 of being found in a female individual. One in every 625 women, or approximately 0.2 per cent of all women, would therefore be expected to be red-green colour-blind; in reality, the figure found is around 0.4 per cent. The reasoning just set out disregarded the influence of consanguineous marriages, which increase the incidence of female red-green colour-blindness. A person with red-green colour blindness does indeed have an increased probability of having a daughter with the same predisposition, since a significant proportion of his first- and second-degree female relatives tend to carry the predisposition in a latent form, and since marriages between first cousins are fairly common. Under these circumstances, the actual figure of 0.4 per cent of women with red-green colour blindness may be regarded as being in good agreement with expectations. Consanguineous marriage has no influence on the frequency of red-green colour blindness in males, since any such trait in males is already expressed in any case (cf. p. 152).

From the pattern of inheritance of colour blindness, we can now also draw conclusions about the inheritance of normal colour vision. It follows from the sex-linked recessive inheritance of red-green colour blindness that certain genetic factors necessary for the development of normal colour vision are sex-linked dominant. These genes, which are thought to be located on the sex chromosome, are therefore never passed from father to son. In a population where red-green colour blindness is overwhelmingly prevalent, the inheritance of normal colour vision could be traced as sex-linked dominant. Furthermore, since total colour blindness is simply recessive, a gene necessary for normal colour vision for blue and yellow must be located on a chromosome other than the sex chromosome and must behave as a simple dominant trait. Thus, the study of populations in which certain genes are absent also sheds light on the inheritance of normal genes.

The hereditary anomalies and disorders of the visual organ discussed here are certainly not the only ones; however, the most common, the best known and the most significant have all been covered. A considerable number of hereditary eye disorders have presumably not yet been recognised as such; this is likely to apply in particular to rare recessive disorders, as even within a large family only a few individuals need to be affected. The discussion of hereditary anomalies of the organ of vision has been kept so detailed because it also served to illustrate more general principles. This will no longer be necessary for the other organ systems.

b) Hereditary disorders of the auricular system.

One of the most serious disorders affecting the ear is the so-called otosclerosis, which manifests itself in increasing hearing loss and very troublesome subjective auditory sensations. Usually from the end of the second decade onwards, hearing declines slowly but steadily until only minimal residual hearing remains, though complete deafness does not usually occur. The inheritance pattern is simple dominant.

Fig. 30.
Excerpt from a family tree showing increasing hearing loss (otosclerosis). According to Körner.

The Pedigree from Hammerslag shows us how the marriage of two hard-of-hearing individuals produced seven offspring, all of whom were in turn hard of hearing . Since the theoretical probability of becoming hard of hearing for each child in this case is three-quarters, it may of course happen that even amongst a larger number of children there is not a single one with normal hearing.

Fig. 31.
Family tree of a family with increasing hearing loss (otosclerosis). According to Hammerschlag.

Apart from otosclerosis, there are certain rarer forms of hereditary progressive hearing loss or deafness, which are caused by atrophy of the auditory nerve or the sensory epithelium in the inner ear. Age-related hearing loss, which occurs particularly early in some families, can also be included in this category.

Fig. 32.
Excerpt from a family tree showing deafness.
According to Dahl.

A proportion of cases of deaf-mutism are caused by a congenital defect of the auditory nerve or by another abnormality of the inner ear. Alongside idiopathic causes of deaf-mutism, parakinetics are, of course, also of great significance. Thus, the inner ear may be destroyed as a result of certain childhood illnesses, e.g. torticollis and scarlet fever. Deaf-mutism also frequently occurs in cases of cretinism, which develops in goitre-prone regions for reasons not yet fully

. Deafness resulting from congenital syphilis usually does not set in until the end of the first decade of life. In Germany, there are around 50,000 deaf-mutes, of whom an estimated quarter owe their condition to pathological hereditary factors. Since the parents of deaf-mute children are generally normal, the trait cannot be dominant; on the contrary, all the evidence suggests that there are recessive factors involved in deaf-mutism.

> In this family, however, an unfavourable clustering has occurred: firstly, because in two successive generations, two individuals in each generation with a latent predisposition to deafness married one another; and secondly, because in both families more than half of the children are deaf, even though the probability of for any single child is only ¼. Five further cases have occurred amongst more distant relatives; yet nowhere within this circle of relatives is the condition found in both parents and children simultaneously. Only when two individuals with the same type of idiotypically caused deaf-mutism marry one another is it to be expected, according to the theory of recessive inheritance, that all their children will also be deaf-mute. This is illustrated by the following family tree:

Fig. 33.
Family tree of a family with deaf-mutism. According to Fay.

> It is, of course, quite natural for deaf-mutes to marry one another; such marriages also occur relatively frequently in the case of other conditions. In Fig. 31, for example, we saw a family depicted in which two hard-of-hearing individuals married one another. In Fig. 33, we see that two marriages in which both parents are deaf-mute produce only deaf-mute children; a third such marriage, by contrast, produced only normal children. This, too, is entirely consistent with the theory of recessive inheritance, provided one assumes that, in this case, the man acquired his condition as a result of an external illness. It should also be borne in mind that there are probably different types of idiopathic deaf-mutism (e.g. one caused by a deficiency of the auditory nerve, another by a malformation of the inner ear). If, then, of two parents, one is deaf-mute as a result of one genetic factor and the other on account of the other, then, in the case of recessive inheritance of both factors, it is likewise not to be expected that the children will be deaf-mute.

It is also consistent with the theory of recessive inheritance that an exceptionally high proportion of deaf-mutes—namely around 7 per cent—are the product of consanguineous marriages, and if only those born deaf are counted, the figure rises to as high as 30 to 40 per cent.

It is also noteworthy that deaf-mutism is much more common amongst the Jewish population than amongst the non-Jewish population. In Berlin, 27 deaf-mutes were counted per 10,000 Jews, compared with only 6 per 10,000 non-Jews. Since it cannot be assumed that deaf-mutism resulting from external illness is so much more common among Jews than among non-Jews, the only conclusion is that the vast majority of cases of deaf-mutism among Jews must be regarded as idiotypical. It follows that the genetic predisposition to deaf-mutism is more widespread in the Jewish population than in the

non-Jewish population, as is also the case with certain other hereditary conditions, although this must not be interpreted as a 'racial predisposition'.

Finally, it should be mentioned that middle ear inflammation (otitis media), which very often results in hearing loss, occurs with a marked familial clustering, as demonstrated, for example, by a study by Stein.

c) Hereditary skin diseases.

Complete and partial albinism, i.e. the lack of pigment in the skin, hair and eyes, has already been discussed above (p. 150). The heritability of freckles or ephelides has also already been mentioned. Belonging more to the realm of pathological conditions are liver f eck and birthmarks (naevi), which result from the formation of organ elements in the wrong place; as a rule, they are more darkly pigmented than the rest of the skin and are often heavily hairy. So-called 'fire marks' (telangiectasias or angiomas), which result from the excessive formation of blood vessels in the skin at localised sites, also occur due to a hereditary predisposition. They often constitute a severe disfigurement and are pathological insofar as they hinder one's progress in life. One form of xanthoma, which manifests as slightly raised patches ranging from yellow to dark yellow in young people, is distinctly hereditary. It is not certain whether the common xanthoma, which is found in older people particularly at the corner of the nose near the upper eyelid, is also hereditary. Occasionally, malignant tumours may develop from birthmarks, arising from the uncontrolled proliferation of certain tissue elements. As a rule, however, all such birthmarks are harmless, because the abnormal growth leading to their formation tends to cease once general growth has been completed.

Neurofibromatosis is also a simple dominant condition, in which numerous nodular tumours develop from the cutaneous nerves. Although the condition is not immediately dangerous, it is extremely troublesome and disfiguring. It could also be classified as a neurological disorder.

In contrast, the so-called xeroderma pigmentosum is highly malignant. Children affected by this condition are born with apparently normal skin; however, under the influence of sunlight, inflammation usually occurs in the first few years of life on exposed areas of skin (face, hands); liver-spot-like pigmentation and scarred indentations develop, and sooner or later cancer tends to develop from this, so that those affected usually die from it within the first or second decade of life and only exceptionally reach the age of 40. Since several children from the same family are relatively often affected by this condition, and since consanguinity is frequently found among the parents, it appears to be a simple recessive predisposition.

Hydraaestivalis is caused by a much less serious sensitivity of the skin to sunlight, in which, following prolonged exposure to short-wavelength light – occasionally even in winter when the sun's rays are reflected off snow – blister-like lesions develop, which may leave scars once they have healed.

Just as sensitivity to sunlight appears to be hereditary, so too does susceptibility to cold. So-called 'frost blisters' (perniões) affect only certain

individuals, and exposure to severe frost is not necessary for the development of frost blisters. It appears to be an anomaly of the cutaneous vascular nerves, similar to that seen in erythromelalgia, in which the extensor surfaces of the hands and feet are intermittently reddened and painful until, eventually, scarring and atrophy of the skin occur. Raynaud's disease, which frequently runs in families and in which, apparently as a result of a disturbance in endocrine secretion (pituitary gland?), vascular spasm in symmetrical areas of the hands or feet can lead to the necrosis of fingers or toes, may also be mentioned here.

The predisposition to acute angioneurotic oedema, in which localised areas suddenly swell up only to subside again after some time, is distinctly hereditary and, indeed, apparently dominant. Dermographism (urticaria factitia) is also caused by an abnormality of the cutaneous vascular nerves; in this condition, the skin swells and reddens in response to slight stimuli, so that one can trace characters with a finger. In the case of so-called hives (urticaria), which manifests itself in the form of numerous small red wheals following the consumption of certain foods such as crabs or strawberries, hereditary predisposition also appears to be a decisive factor. The predisposition to erythema exsudativum multiforme is clearly hereditary; this condition bears a certain resemblance to urticaria but is accompanied by inflammation and blistering, particularly on the extensor surfaces of the hands, and infectious factors appear to play a significant role in its onset. It is noteworthy that the same individuals are often affected by rheumatoid arthritis and endocarditis either concurrently with their skin condition or at other times. It is therefore a hereditary predisposition that affects a wide variety of organs.

The group of cutaneous vascular anomalies discussed here is closely related to what is known as exudative diathesis (see p. 192 below), a constitutional abnormality that manifests itself particularly in the early years of life, including, amongst other things, a more or less transient nodular, itchy rash (strophulus). This category also includes prurigo, a severely itchy nodular rash that occurs particularly on the extensor surfaces of the arms and legs and persists to a greater or lesser extent throughout one's life. Pruritus occurs without such a rash; it is a very troublesome itching that intensifies periodically and, particularly in old age, often reaches an excruciating intensity. All these abnormalities of the cutaneous nerves could also be discussed in the chapter on hereditary nervous disorders or in the one on

constitutional abnormalities. Essentially, every genetic factor exerts an influence on all organs. It is therefore not the case that a single genetic unit corresponds to a very specific part of the body (such as a specific group of cells within an organ), as some researchers previously believed; rather, the totality of genetic units, in interaction with external conditions, determines the body's overall constitution. This is entirely consistent with the fact that a particular genetic trait may manifest itself predominantly in a specific organ, or that a genetic trait, despite having a more general manifestation, may acquire pathological significance primarily in a specific organ, as is the case, for example, with the albinism trait affecting the eye.

Hereditary chronic oedema of the legs is also caused by a pathological condition of the vascular nerves; several fairly large family trees with dominant inheritance have been documented in connection with this condition. Inflammatory symptoms affecting the vascular nerves lead to a slowly progressive swelling of the legs, which can eventually become so severe that movement is no longer possible.

Epidermolysis bullosa is a distinctly hereditary condition of the skin in which blisters form in response to even slight stimuli such as pressure, impact and, in particular, friction. Although the condition is not directly dangerous, it is very troublesome and severely impairs the ability to work. Different forms are observed in different families. The trait is generally dominant; however, skipping a generation does occasionally occur. It therefore appears that the trait can be suppressed by certain other traits, which, admittedly, are present only in exceptional cases. Under these circumstances, the ratio of 180 affected to 209 unaffected siblings, as found in 27 family trees, corresponds well with the theoretical expectation of 1:1 for dominant inheritance.

Fig. 34.
Family tree showing epidermolysis bullosa. According to Colombini.

One must bear that even that individual of classified as Pemphigus vulgaris malignant bullous skin conditions may perhaps be hereditary—certainly not dominant, but possibly recessive—as suggested, for example, by reports that the parents of

patients are more often blood relatives and that pemphigus occurs more frequently than usual in the Jewish population.

The skin's resistance to external stimuli varies greatly from person to person. If inflammatory changes develop on the skin under the influence of mild chemical stimuli, accompanied either by exudation or by the formation of scabs and pus, this is referred to as eczema. However, different individuals are affected by eczema to very different degrees under the same external influences. For some, even brief contact with tar oils can trigger eczema, whilst others remain free of it despite prolonged exposure. There are, in fact, 'eczema families' in which certain members suffer repeatedly from eczema, which often occurs even without any detectable external causes. In hereditary diabetes, protracted eczema of the hands and feet occurs; similarly, this is seen in constitutional achylia (deficient production of gastric juice), in gout and in other constitutional disorders. In so-called constitutional infantile eczema, the idiotypical nature of the condition is particularly evident, as external irritants which lead to eczema later in life do not yet play a role during infancy.

Related to the predisposition to eczema is so-called seborrhoea, a constitutional anomaly associated with excessive activity of the sebaceous glands. Seborrhoea, which usually runs in families, often leads to hair loss and baldness. Acne vulgaris is also caused by excessive and pathologically altered activity of the sebaceous glands; it is a troublesome, albeit harmless, condition of puberty in which numerous sebaceous plugs (so-called blackheads) develop in the hair follicles on the face, the nape of the neck, the back and the chest, where numerous sebaceous plugs develop in the hair follicles (so-called blackheads), which often turn into pustules accompanied by pus formation. Acne tends to run in families; it often appears to be a sign of constitutional weakness, particularly digestive weakness.

So-called 'scaly eczema' or psoriasis also frequently runs in families. This common condition usually manifests as numerous well-defined, reddish-brown areas of inflammation, covered with coarse scales, on the extensor surfaces of the arms and legs. Although those suffering from psoriasis may often remain symptom-free for years between individual flare-ups of the disease, on the whole the condition must be regarded as incurable. In severe cases, almost the entire body may be covered with inflammatory lesions. Judging by the nature and spread of the lesions, it appears that the individual outbreaks of the condition are caused by microscopic organisms.

Psoriasis is therefore presumably due to a hereditary susceptibility to certain as yet unknown parasites.

Something similar appears to apply to so-called lichen ruber, a chronic skin disease characterised by the formation of clusters of red nodules, of which there are both benign and malignant forms. The condition appears to be caused by an infection to which only certain families are susceptible.

So-called sweaty feet and sweaty hands are caused by an abnormality in sweat gland activity; the predisposition appears to be simply dominant. In addition to excessive sweating (hyperhidrosis), which is often accompanied by the production of foul-smelling sweat (bromhidrosis), insufficient sweating (oligidrosis) and a tendency for blisters to form on the fingers and toes due to sweat (dyshidrosis) also appear to be idiopathic in nature. A complete absence of sweat gland function (anhidrosis) also occurs as a hereditary anomaly. In affected individuals, hair and teeth are also stunted.

Hereditary abnormalities of the skin's keratinous structures—which also include nails and hair—are particularly common. In hereditary keratosis (keratoma, tylosis), the skin on the soles of the feet and palms of the hands becomes horny and brittle shortly after birth; externally, the area of keratinisation is demarcated from the normal skin by a blue-red border. The condition, which has occasionally been traced through five generations of a family, is simply dominant.

Fig. 35.
Excerpt from a family tree showing keratosis according to Thost.

What is remarkable about this family tree is that a man affected by keratosis fathered affected children with two different healthy women, thereby highlighting the dominant pattern of inheritance particularly clearly.

In hereditary ichthyosis ('fish-scale skin'), the entire surface of the skin is covered with more or less coarse horny plaques. There are several forms. In the mildest form, the skin shows only a flaky, dry appearance. In a more severe form, the skin is covered with actual horny plaques, giving it the appearance of crocodile skin ('sauriasis'). In an even more severe form, the horny masses protrude as pointed, dark growths (ichthyosis hystrix, porcupine skin). The milder forms are apparently dominant, whilst others appear to be recessive, such as ichthyosis foetalis or keratosis universalis, which refers to a condition affecting newborn, usually immature infants who are completely covered in horny masses and are not viable.

In the case of dominantly inherited porokeratosis, brownish raised lesions develop on the extensor surfaces of the hands and feet; these reach the size of a lens or a coin and are bounded on the outside by a

keratinous rim.

Keratosis pilaris is also hereditary; in this condition, horny plugs develop within the hair follicles on the extensor surfaces of the arms and legs, and in more severe cases also on the back, causing the hair to eventually fall out and resulting in numerous small scars.

P s o r o s p e r m o s i s v e g e t a n s begins with the formation of yellowish-brown, shiny raised lesions, which eventually soften and give off a very foul odour. The nails also become thickened, brittle and discoloured. The condition occurs in clusters within families; however, the mode of inheritance does not appear to be dominant.

In the case of Aplasia pilorum moniliformis (Monilithrix, spindle hair), children are born with normal hair. However, only sparse, short hairs subsequently grow back; these have colourless, thin patches at regular intervals of about 1 mm, at which points they tend to break off not far from the root. The condition is simply dominant.

The condition known as Trichorrhexis nodosa, in which the hair breaks off at knotty, swollen points, also appears to be hereditary.

Baldness can be caused by various causes, e.g. as a result of seborrhoea. In many families, baldness occurs in several male members at roughly the same age. In other families, the hair on the head remains largely intact into old age. The onset of greying also occurs earlier or later, depending on hereditary predisposition.

Fig. 36.
Family tree showing aplasia pilorum moniliformis (abridged) according to Sabouraud.

General hair loss (hypotrichosis, alopecia) also occurs on a hereditary basis. In those affected, the scalp bears only a thin, soft down from youth onwards; in men, the beard is indicated only by a few stubble-like hairs; the rest of the body hair is completely absent. The nails are thick and misshapen, and the teeth are stunted. The condition is dominant.

On the other hand, there are also families of 'hairy people'. This abnormal abundance of hair (hypertrichosis), which is also accompanied by defective tooth formation, is due to the persistence of the foetal hair coat (lanugo).

Complete absence of nails (anonychia) appears to be a dominant condition. Poor attachment of the nails to the nail bed, longitudinal ridging of the nails and abnormal brittleness of the nails

occur on an idiotypic basis. Also hereditary is that anomaly of the nails in which the skin margin at the nail root does not detach properly from the nail, which tends to lead to troublesome tears. This anomaly, which is usually accompanied by abnormal softness and small size of the nails, appears to be dominant. White spots on the nails (leukonychia) also occur with increased frequency in certain families. In cases of severe leukonychia, where the entire central part of the nail is white in colour, dominant inheritance has been observed. In one family, severe leukonychia was observed in 19 members, and 17 of these also had atheromas (see below). That peculiar, pronounced longitudinal curvature of the nails, which is often associated with thickening of the distal phalanges, also occurs with increased frequency within families. These
'clubbed fingers' often develop as a result of lung conditions, but also seem to be associated more frequently with a mere predisposition to them.

In onychogryphosis, the nails are severely thickened and curved like claws; these claws can grow up to 20 cm long and up to 2 cm thick. In one family, uninterrupted inheritance through four generations on the female line was observed.

So-called atheromas, or 'grützbeutel', result from invaginations of skin folds and are usually found amongst the hair on the head, where they can reach the size of a hen's egg or larger. This growth occurs because the secretions and waste products of the skin (sebum, keratinous scales, hairs) gradually accumulate, as they have no way of escaping. Atheromas usually occur in multiple instances in the same person and, furthermore, in several members of the same family. The predisposition appears to be simply dominant, but probably does not manifest itself in every case.

Fig. 37.
Family tree showing bullous dystrophy. According to Mende da Costa.

A generalised condition manifesting predominantly on the skin is bullous dystrophy. This consists of congenital hairlessness, blistering and atrophy of the skin, patchy depigmentation and pigment accumulation in other areas, and stunted growth of the head and fingers. The condition is probably caused by an internal glandular disorder (pituitary gland?). This rare condition is X-linked recessive, although this pattern of inheritance is not known to occur in any other skin condition.

d) Malformations.

In a certain sense, all hereditary conditions can be regarded as malformations. In most cases, however, the malformation is not readily apparent externally; rather, it affects the internal structure of the organs or their smallest components, the cells. The abnormal structure of the cells results in abnormal organ function. Usually, however, the term 'malformation' does not refer to the pathological formation of many small organ components, but rather to conspicuous deviations in external form.

Every hereditary malformation stems from a malformation of the genetic material, which is, however, not directly perceptible. An unbroken developmental sequence extends from the malformation of the genetic material in the fertilised egg right through to the malformation present in the newborn. Strictly speaking, therefore, there is no specific point in time at which a hereditary malformation arises; at most, there is the point at which it first becomes perceptible. It is customary to refer to only those deviations in form as malformations which are already fully formed at birth. In principle, however, even birth does not mark the final point at which malformations can become apparent. After all, individual development is by no means complete at birth. One could therefore also describe the abnormal length of the eyeball in cases of severe short-sightedness as a malformation, even though it is not present at birth but only develops later. The approximate formation of the external shape and the individual limbs is recognisable not only around the time of birth but as early as the third month of gestation; the same applies, therefore, to most malformations.

In addition to hereditary malformations, there are also those of a paratypical nature, which arise as a result of the mother's illness, toxic effects or mechanical influences. Defects in the structure of the amniotic membranes, particularly constrictions of the amnion (the so-called 'sheepskin') and excessive tightness of the amniotic membranes combined with a lack of amniotic fluid, can lead to serious developmental disorders; these, however, are naturally not hereditary. Only if the constriction or cord-like formations of the amnion were themselves hereditary could heredity play a role here; in that case, however, the malformations in the offspring would not correspond in form to those of their ancestors, but would vary more or less irregularly. Amniotic cords can cause constriction of limbs, with the result that the children in question are then born, for example, with only one arm or only one foot. In general, however, the significance of amniotic damage is certainly not nearly as great as was long believed. There is scarcely a malformation that has not already been attributed to this cause, including numerous cases whose hereditary nature is beyond doubt. If a malformation occurs in the same form in several members of a family, it can be said that it is certainly not caused by the amnion. The symmetry of malformations also strongly argues against this. If, for example, a malformation is found in the same way on both hands, one may conclude that it is, in all probability, idiotypical in origin; the same applies to malformations found on the feet in a similar manner to those on the hands.

Malformations can be defined as deviations in the structure of the body, particularly in its external form, which arise from faulty development and which impair the

the organism's ability to adapt. We are concerned here only with those caused by idiotypy. Congenital absence of fingers due to constriction by amniotic cords is much more akin to the loss of fingers through injury later in life than to the absence of fingers due to hereditary monodactyly. Naturally, mutilations resulting from amniotic cord constriction cannot be passed on to subsequent generations any more than those caused by external force later in life. The better-known hereditary malformations of the limbs are, for the most part, dominant.

The best-known hereditary malformation is probably polydactyly, in which supernumerary fingers are present in a more or less complete form. In particular, six-fingeredness has been traced through several generations in a fairly large number of families. In a village in southern France (Izeaux, Department of Isère), the anomaly has become so widespread that the majority of all inhabitants are said to have been affected by it. In most families, members with six fingers also have six toes. However, the anomaly is also said to occur separately on either the hands or the feet.

Fig. 38.
Family tree of a family with polydactyly. According to Lukas (excerpt).

A much rarer but also more severe malformation is hereditary ectodactyly (absence of fingers), which can extend to the point of having only one finger. The condition also appears to be simply dominant, though the degree of finger deficiency can vary greatly even within the same family.

A distinctly dominant condition is syndactyly, in which two or more fingers are fused to ly the third and fourth. There is one form in which the fingers are connected only by a kind of web, and another in which the bones are also fused. Similar anomalies also occur in the feet.

Symphalangy (orthodactyly) is caused by the fusion of bone segments within the same finger. Most commonly, the middle joints of the middle fingers are missing. Consequently, the hand

consequently not be closed. Various forms have been observed in different families, all of which appear to be simply dominant; yet even within the same family the degree of the condition can vary considerably, with sometimes more and sometimes fewer joints missing.

In c l i n o d a c t y l i a, which is also dominant, the joints of the nail phalanges are ossified. In c a m p t o d a c t y l i a, the little finger and usually the ring finger cannot be extended; this condition is also dominant. The so-called Dupuytren's contracture bears an external resemblance to these conditions; it is a disorder that develops over the course of a person's life and consists of a contraction and stiffening of the tendons and ligaments on the palm side of the hand, particularly towards the little finger. The condition, which occurs predominantly in men, has been observed to run through several generations in some families. The predisposition therefore appears to be simply dominant; occupational hazards evidently contribute to its development. There appears to be a certain association with chronic joint changes (deforming arthritis).

Fig. 39.
Family tree of a family with brachydactyly. After Farabee (excerpt).

Short-fingeredness, or brachydactyly, is also simply dominant; in this condition, the fingers are not only greatly shortened but also have only two phalanges instead of three, whilst the thumb has only one instead of two. At the same time, the length of the arms and legs—and indeed that of the entire body—is reduced. Men with brachydactyly are found to be, on average, 21 cm shorter, and women 12 cm shorter, than their normal siblings. The dominant trait therefore inhibits the longitudinal growth of the bones in general.

It is of some historical interest that brachydactyly was the first instance in which the validity of Mendel's laws for humans was confirmed. F a r a b e e found 36 short-fingered siblings to 33 normal ones in one family, 42 short-fingered to 33 normal in another, and 21 to 26 in a third; together, this amounts to 99 to 92, which corresponds very closely to the ratio of 1:1.

Quite similar is the equally dominant m i n o r b r a c h y d a c t y l i a, in which the finger phalanges are present in normal numbers but are very short.

The so-called 'split foot' is a hereditary malformation in which the feet taper forwards into two separate parts, each bearing a single toe. A hereditary 'split hand', in which the hand is split almost to the base, also occurs.

'Clubfoot' (Pes varus) refers to a club-like deformity of the feet, in which they are severely turned inwards, so that the soles face each other and the foot touches the ground only with its outer edge. At the same time, the tip of the foot is usually turned downwards. Almost one in every 1,000 newborns is affected by clubfoot; male children are affected about twice as often as females; in about half of the cases, both feet are affected. According to Fetscher, the predisposition to clubfoot appears to be recessive; in any case, it is not dominant. However, even in the case of homozygous inheritance, the genetic predisposition appears to lead to a corresponding developmental disorder only in a proportion of cases. An unfavourable position within the uterus may be a contributing factor, for example where the amniotic sac is very constricted. Malformations of the spinal cord are often found at the same time, suggesting that a malformation of the nerves may be the cause of the clubfoot.

Fig. 40.
Family tree of a family with clubfoot according to Fetscher.

I am indebted for the above family tree to the kindness of Dr Fetscher in Tübingen, who permitted me to reproduce this family tree from one of his as yet unpublished works. The child with clubfoot in the third generation was the result of incest between a brother and sister, an example of how recessive traits can become homozygous through inbreeding.

Likewise, that outward curvature of the foot, which is accompanied by a flattening of the arch and is known as a flat foot, appears in most cases to be based on a hereditary predisposition and to be brought about by external influences (such as prolonged standing). The outward bending of the big toe, known as hallux valgus—which was previously attributed solely to the pressure of pointed shoes—also appears to result from an interaction between hereditary predisposition and external influences. The so-called X- and O-shaped legs are also likely to result from an interaction of hereditary and external factors.

Hereditary predisposition is of decisive importance in congenital hip dislocation (Luxatio coxae connata), a by no means rare condition in which the head of the femur does not sit within the hip socket. The condition is found about seven times more frequently in females than in males, which is related to the fact

the fact that, even in healthy females, the femur is positioned at a steeper angle to the pelvis, meaning that, in the presence of a pathological predisposition, the head can slip out more easily. It is not possible to trace the inheritance pattern continuously through the generations; instead, as a rule, only a few siblings are found to be affected by the condition. More often than not, the predisposition develops on only one side.

As a rare hereditary malformation occurs a deficiency of the kneecaps, which appears to be simply dominant.

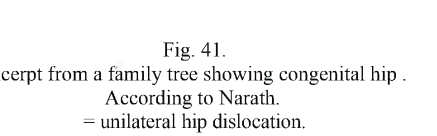

Fig. 41.
Excerpt from a family tree showing congenital hip .
According to Narath.
= unilateral hip dislocation.

Hereditary malformations of the limbs also include multiple enchondromas and exostoses (cartilage and bone tumours), although they are not yet present at birth but only develop over the course of childhood into lumps of varying sizes, which can severely disfigure the hands in particular and render them unusable. As the skeleton stops growing, so too does the growth of these tumours; this highlights the deformity-inducing nature of the condition. It has been observed across several generations; the predisposition therefore appears to be simply dominant.

At this point, mention should also be made of a rare malformation known as dysostosis cleidocranialis. In this condition, the skull is markedly widened, whilst the growth of the facial bones is stunted. The bones of the shoulder girdle are completely atrophied; the clavicles may be entirely absent, allowing the shoulders to be brought together at the front. Milder forms of this developmental disorder are occasionally found in family members. The condition is certainly idiopathic in nature, but the pattern of inheritance has not yet been clarified.

In the region of the genital organs, a distinctly hereditary malformation is the so-called hypospadias. In men affected by this condition, the opening of the urethra is not located at the tip of the penis, but on the underside, situated more or less far towards the base. It is estimated that approximately one in every 300 males is affected by this to a greater or lesser degree. The condition has been traced through several generations along the male line in some families. Naturally, it cannot manifest itself in females, but it can be passed on to male children even by women who are themselves healthy. Hereditary traits that manifest themselves in only one sex do occur occasionally in other contexts as well. This mode of inheritance must not be confused with sex-linked recessive inheritance (see p. 152), as has often been the case. Even outwardly, the mode of inheritance of a dominant trait—which, however, cannot manifest itself in the female sex—differs from that of a sex-linked

recessive pattern in that, in the latter case, the trait cannot be passed from father to son, whereas in the former it can.

> In cases of severe hypospadias, the penis may be very small and the scrotum split into two halves, giving the appearance of hermaphroditism. Since sexual differentiation in humans is also idiotypically determined, it may be assumed that even the — albeit very rare — cases of true hermaphroditism in humans are based on idiotypic factors. The term 'true hermaphroditism' is used when both male and female gonads are present side by side. If only male or only female gonads are present, but in conjunction with external characteristics of the opposite sex, the terms 'pseudohermaphroditism masculinus' or 'pseudohermaphroditism femininus' are used. Those forms of pseudo-hermaphroditism in which male and female sexual characteristics are present in varying combinations are certainly idiotypical in nature. In animals, such hermaphrodites can be artificially produced through certain cross-breeding programmes, for example by crossing the Japanese silkworm with the European silkworm. In human families, pseudohermaphroditism has occasionally been observed among siblings. Male pseudohermaphroditism is said to be much more common than the female form; as a rule, however, this is likely to be the aforementioned severe hypospadias, which can hardly be classified as pseudohermaphroditism.
>
> A relatively harmless hereditary malformation is the abnormal tightness of the foreskin in males, known as phimosis, which is often accompanied by abnormal length.
>
> Among the malformations of the female reproductive organs, the not infrequent structural abnormalities of the uterus (duplications, etc.) are likely to be largely idiotypic in nature, as is the underdevelopment of the reproductive organs in general (cf. infantilism, p. 191). The occurrence of supernumerary mammary glands (hypermastia) or simply supernumerary nipples (hyperthelia) can also be counted among idiotypical malformations. Hyperthelia has been observed across several generations of a family. The presence of developed mammary glands in males can be classified as pseudohermaphroditism.

If the male gonads do not descend into the scrotum in the normal manner before birth, but remain in the abdominal cavity or the inguinal canal, this is referred to as cryptorchidism. This occurs in conjunction with hypospadias, but more frequently on its own. Hereditary predisposition certainly plays a significant role in its development. Most commonly, only one of the two testicles remains in the abdominal cavity or the inguinal canal. This condition cannot be regarded as entirely harmless, because an inguinal testicle cannot develop normally but is exposed to pressure-related damage and inflammation. Abnormal smallness or the complete absence of the testicles is also likely to be idiopathic in nature, although no reliable information is available on this.

In connection with disorders of testicular descent, consideration should also be given here to the predisposition to inguinal hernias, which are

largely result from insufficient closure of the inguinal canal through which the testicle has descended. An actual hernia usually develops only gradually under external influences, particularly during exertion such as lifting, etc., whereby parts of the peritoneum and, in most cases, the intestine protrude through the inguinal canal. Approximately one in every 20 to 30 men suffers from a hernia, whilst among women the incidence is around 1 in 150. Hernias represent a significant impediment to the ability to work and, on the other hand, can also pose a serious threat to life through incarceration and inflammation. The question of whether a predisposition to hernias is hereditary is therefore of the utmost practical importance. In some families, a cluster of inguinal hernias is unmistakable, and one gets the impression that there are dominant predispositions to them, which, however (similar to those for hypospadias), do not tend to manifest themselves in females. Taking the whole situation into account, it must be assumed that heredity plays a role in the development of inguinal hernias that is no less significant than that of external causes.

> Hereditary predisposition is also likely to play a role in the development of so-called epispadias, a rare cleft on the upper surface of the penis, which can extend as far as a cleft in the anterior abdominal wall and the bladder. Since children born with this condition do not survive, dominant inheritance is, of course, out of the question.

In the region of the face and jaws, cleft lip and palate are not uncommon and are often due to hereditary predisposition. The so-called 'hare lip' consists of an indentation or cleft in the upper lip to the side of the midline. In more severe cases, the cleft may also extend to the bony jaw; and in the most severe cases, the palate may also be cleft; this is then referred to — albeit a rather uninformative term — as 'cleft palate'. These clefts, like many other malformations, are due to developmental abnormalities; they arise when the recesses between the upper jaw and the premaxilla, present at an early stage of development, fail to close completely. It appears that a single predisposition can, depending on other conditions during development, result in varying degrees of severity of these developmental abnormalities. The occasional skipping of a generation suggests that the predisposition often need not manifest itself at all.

With regard to hereditary anomalies affecting the jaws, it should also be noted that the lateral incisors may frequently be absent in several members of a family. In other families, the so-called wisdom teeth (the third molars) never appear. An abnormally wide gap between the central incisors ('trema') appears to be dominantly inherited. Overcrowding of the teeth appears to be caused more often by a combination of a hereditary predisposition to large teeth and a predisposition to small jaws. Hereditary dental anomalies associated with malformations of the coat have already been mentioned above.

Fig. 42.
Pedigree of a family with a hares' lip. After Rayley (abridged). In the affected individual of the second generation, the cleft also extended to the palate.

The protrusion of the lower jaw is also hereditary (mandibular prognathism, prognathism). This apparently dominant anomaly is more pronounced in males, who normally have a more strongly developed lower jaw. Prognathism, which has been traced through many generations in the Habsburg family, is does not, however, strictly speaking, count as a malformation. Abnormal smallness of the lower jaw also occurs on a hereditary basis.

A harmless hereditary anomaly is the so-called fused earlobe; in more severe cases, the earlobe is entirely absent. People have sought to interpret fused earlobes as a sign of degeneration and believed them to be particularly common amongst criminals and the mentally ill. However, they are found just as frequently amongst normal people and in perfectly healthy families. It is evidently due to a simple hereditary trait which is independent of other Mendelian traits and which therefore allows no conclusions to be drawn about the rest of the physical or mental constitution.

The situation is quite different when it comes to certain more severe malformations of the skull and brain. For instance, an abnormally small skull and, consequently, an abnormally small brain (microcephaly), which often occurs in clusters within families, is associated with congenital mental retardation or idiocy. In one reported case, five of a mother's children were found to have microcephaly with mental retardation.

Other congenital malformations include the complete absence of the head (acrania) or of the brain (anencephaly). As the affected foetuses are, of course, not viable, the hereditary nature of such malformations has hitherto almost never been considered, but wrongly so, as I need hardly elaborate.

In the region of the spinal column and the spinal cord , developmental abnormalities occur in the form of clefts, which often manifest as fracture-like protrusions in the lower part of the spine (spina bifida). Here too, presumably hereditary predispositions, alongside parakinetics, play a role.

Fig. 43.
Excerpt from a family tree showing spinal curvatures (scoliosis) according to Klassen.

Heredity has a significant influence on the development of lateral curvatures of the spine, known as scoliosis. Since scoliosis can be traced through several generations in some families, both on the paternal and maternal sides, there appear to be simply dominant genetic predispositions for it. However, external factors are also of great significance in the development of scoliosis, e.g. rickets.

Funnel chest is a deformity in which the lower end of the sternum is deeply indented, resulting in a funnel-shaped hollow in the anterior chest wall. There appears to be a dominant genetic predisposition to this condition. Occasionally, a similar condition may also arise as a result of pressure, for example, in shoemakers.

Fig. 44.
Family tree of the family with funnel chest. After Paulsen.

The type of dwarfism known as chondrodystrophy or achondroplasia can also be regarded as a malformation. In this condition, the cartilage zones within the bones – where longitudinal bone growth takes place – are poorly developed. Affected individuals therefore have stunted, short limbs (micromelia), whilst the head is of approximately normal size. There are various forms of chondrodystrophy of differing severity. The milder forms are referred to as chondrohypoplasia. Brachydactyly can be regarded as a very mild form, in which the limbs are also relatively short. There also appear to be more pronounced forms of chondrodystrophy which, like brachydactyly, are simply dominant. Women with chondrodystrophy, however, have pelvises so narrow that natural childbirth is usually not possible. The most severe forms of chondrodystrophy, which are observed in children who die whilst still in the womb, are certainly not dominant. One must consider a recessive pattern of inheritance for these conditions.

Fig. 45.
A family with chondrodystrophy. According to Nizhoff.

Following on from the discussion of malformations, we should also consider here the extent to which hereditary predisposition plays a role in the formation of twins. Identical twins arise as a result of the complete division of a fertilised egg into two halves, each of which then develops into a child. If this separation is not entirely complete, but both children are fused together—which, however, happens only very rarely—the malformation becomes clearly apparent. The formation of fraternal twins, which arise from two different eggs fertilised simultaneously, cannot be regarded as a malformation. Since identical twins are always of the same sex, whilst

fraternal twins exhibit the usual sex distribution, it is possible to deduce the frequency of identical twins from the statistically observed distribution of the twins' sexes. This shows that approximately a quarter of all twins are identical. Whilst twin births generally occur in about one in 80 births, the birth of identical twins occurs in one in 300 to 350 births. Hereditary factors certainly play a role in the occurrence of twins, as they do in multiple births in general. Mothers and daughters of mothers who have given birth to multiples show a higher rate of multiple births than the average. However, multiple births recur in only 5–6 per cent of mothers who have given birth to multiples; the relevant genetic predispositions therefore come into play only relatively rarely. Naturally, the predisposition to identical twins is of a completely different nature to that for fraternal twins. However, no further details are known on this matter.

Situs viscorum inversus, a condition in which the positions of all the organs are reversed between right and left, is occasionally found in siblings. Four cases of familial occurrence have been described in the literature.

e) Heritable constitutional anomalies.

The concept of a 'constitutional anomaly' is just as difficult to define precisely as that of a 'malformation'; nevertheless, like the latter, it is very useful in practical terms. One speaks of a 'strong' or 'weak' constitution, thereby referring to the degree of general resistance to harmful influences, exertion and disease. In many cases, the constitution is already evident in one's outward appearance, in one's habitus. Constitutional anomalies which manifest themselves less in the habitus than in certain functional peculiarities—and which are therefore primarily rooted in the chemical and physiological nature of the tissues—are usually referred to as diatheses.

> Constitutional anomalies form a continuum with malformations, metabolic disorders, disorders of the endocrine glands and susceptibilities to specific diseases. The concept of constitution, like that of disease, is oriented towards the probability of persistence. However, whilst the concept of disease already presupposes a relatively low probability of persistence, the concept of constitution is indifferent in this respect. One also speaks of a healthy constitution. It is not appropriate to restrict the concept of constitution to hereditary predispositions, as Tandler and, along with him, a few others would have it. This does not correspond to common usage, from which there is no reason to depart here. Cretinism, for example, is generally classified as a constitutional anomaly; however, it has not been proven—nor is it even probable—that it is idiotypically determined. We therefore use the term 'constitution' quite generally to denote the physical constitution in terms of its likelihood of survival or, which amounts to the same thing, its resilience. If a susceptibility relates only to individual diseases, one does not speak of a constitutional weakness, but of 'disposition'.
>
> In the previous chapters, several hereditary anomalies have already been discussed which, given the fluid boundaries of the term, could also be classified as constitutional anomalies. For example, the blue-grey colour of the sclera of the eye (the 'white' of the eye) is a manifestation of a general constitutional disorder, idiotypic bone fragility or osteopsathyrosis, in which bone formation is inadequate from the outset, so that spongy tissue develops instead of solid bone. The children affected are often born with fractures; and later in life, fractures occur repeatedly even as a result of minor incidents. This rare condition appears to be dominant.

The prime example of a constitutional anomaly is so-called asthenia or the asthenic constitution. Those affected are of a slight build; the chest is narrow and usually flat, with a relatively long torso. The heart is usually small

and weak, and hangs down, as it were, within the frail chest. It is due to this slender build that the stomach and other abdominal organs also sag. The musculature, too, is usually weak and flaccid. The spine is not held upright, resulting in 'poor posture', which is therefore less a consequence of carelessness than of laxity in the intervertebral joints and the back muscles. The asthenic individual tires easily, both from physical and mental exertion. The laxity of the digestive organs impairs nutrition. This is also linked to the anaemia that usually occurs at the same time, though this is less a matter of the blood being too diluted than of there being too little of it overall.

Owing to their weak physical constitution, asthenics cannot offer the same level of resistance to all manner of diseases as people of normal build; and since tuberculosis plays a particularly significant role amongst the diseases affecting our population, asthenics succumb to consumption in relatively large numbers.

Owing to its high prevalence and its extraordinary significance for health and physical capacity, the asthenic constitution is one of the most important anomalies of all. So-called general physical weakness, which constitutes the most common reason for unfitness for military service and which is assessed primarily on the basis of the ratio of chest circumference to body height, is usually a manifestation of asthenia. It has even been described as a hypoplastic (underdeveloped) constitution, and certainly with some justification, at least in part. This is particularly so as the gonads of asthenic individuals are often underdeveloped.

The causes of asthenia are certainly essentially idiotypical in nature; it occurs with increased frequency in many families. The asthenic physique tends to become more pronounced only from the beginning of the second decade of life. Apart from hereditary factors, environmental influences are certainly also significant in the development of asthenia. For instance, an urban lifestyle and, in particular, prolonged sitting during the formative years – as is the case, for example, with our education system – contribute to the development of a frail physique. Of course, one must not overlook the fact that idiopathic weakness of the spine, as well as abnormal fatigue in general, is often accompanied by an aversion to physical exercise. Thus, one factor reinforces the other.

Attempts have been made to attribute the development of asthenia to a disturbance in the so-called endocrine glands, that is, those organs responsible for the internal metabolism of bodily fluids. However, the failure or dysfunction of any one of these glands results in very specific changes that are not consistent with the asthenic constitution. Since some researchers tend to attribute every constitutional anomaly to disturbances in internal secretion, a 'polyglandular theory' has been put forward in place of a 'uniglandular' one; that is to say, all the endocrine glands are said to be involved, to a greater or lesser extent, in the development of asthenia. This may well be true to a certain extent; but precisely for that reason, the decisive cause of asthenia cannot lie there; for then the question arises all the more: how is it that so many organs fail to function properly at the same time?

Stiller, who was the first to provide a comprehensive description of the clinical picture of asthenia, rightly classified it as a distinct disease. This has been criticised on the grounds that a constitutional anomaly does not in itself constitute a disease. On closer reflection, however, it becomes clear that 'Stiller's disease' certainly fulfils the definition of a disease. It is a condition at the limits of one's capacity for adaptation, which causes its sufferers manifold subjective distress and compels them to exhibit all manner of abnormal reactions. The term 'disease' is still often thought of too narrowly in terms of infectious diseases and other conditions with predominantly external causes (cf. p. 145).

Asthenia is thus a clinical syndrome, but not an idiotypical entity. There are probably several different genetic predispositions that can cause asthenia; nor is it unlikely that more severe forms may be caused by the interaction of several different genetic predispositions, and that, depending on the nature and number of the contributing genetic predispositions, milder and more severe degrees of asthenia may arise. In some families, there appear to be simply dominant genetic predispositions to asthenia.

Fig. 46.
Family tree of a family with a 'habitus asthenicus'. After Paulsen.

Closely related to asthenia is the I n f a n t i l i s m, a constitutional anomaly that manifests itself as a failure to progress at childhood stage of development. A clear distinction between the two conditions is not possible. Some doctors prefer to use the term 'asthenic infantilism'. In individuals with infantilism, the sexual organs remain small and underdeveloped. Infantilism is, after gonorrhoea, probably the most common cause of female infertility. If pregnancy does occur, it relatively often ends in miscarriage, because the foetus cannot develop properly in the underdeveloped uterus. Infantilism is also evident in physical appearance. Infantile girls often still have almost childlike facial features even at the age of 25 or 30, which gives them a kind of false youthfulness. Mentally, too, adults

exhibit childlike traits; they are easily influenced by immediate sensory impressions and experiences, as well as by the will of others. They have little energy, are ill-suited to serious work and tend towards playful activities.

As regards the causes of infantilism, childhood tuberculosis is frequently regarded as the cause; it is more likely, however, that that peculiar form of childhood tuberculosis, often referred to as scrofula, is itself a consequence of a hereditary anomaly. Very often, in fact, infantilism is accompanied by an abnormality of the lymphatic system, which constitutes a predisposition to the development of glandular tuberculosis.

This abnormal development of the lymphatic system also frequently occurs without the other features of infantilism. This is then referred to as S t a t u s l y m p h a t i c u s (lymphatic diathesis, lymphatism). Since the lymphatic system is more highly developed in normal children than in adults, lymphatic diathesis has been regarded as an excessive manifestation of a characteristic of childhood.

> It has also been suggested that this is linked to an abnormal persistence of the thymus gland beyond childhood, and attempts have been made to attribute deaths occurring for trivial reasons—such as minor operations—or without any apparent cause, as well as psychological abnormalities and a tendency towards suicide, to this condition. However, recent findings suggest that this view is untenable. The fact that the thymus gland is found to be fully intact in young people who have died suddenly, but not in those who have died after a prolonged illness, seems simply to be due to the fact that, in the case of prolonged illness, the thymus gland atrophies particularly severely.
>
> If the clinical picture is characterised by enlargement of the lymphatic structures of the throat and, in particular, the tonsils, one tends to speak of an adenoid constitution. The resulting difficulty in nasal breathing has also been attributed to all manner of disorders, hearing loss and delayed mental development. As the children in question tend to breathe through their mouths, the upper jaw is said to become narrow, and the palate narrow and pointed. It is far more likely that the narrow palate is a direct manifestation of the same hereditary narrowness of the upper jaw, which is also evident in the narrowness of the nasopharynx.

Status lymphaticus usually only develops in the second half of the first decade of life. In infancy and early childhood, it is often preceded by the symptoms of what is known as exudative diathesis. These infants are prone to sores; scales and crusts form on the head, the tongue shows whitish spots and lines, and the so-called ‘milk crust’ appears on the cheeks.

At the same time, catarrhal and asthmatic symptoms frequently develop. Exudative diathesis is often accompanied by abnormalities in nutrition and the nervous system. However, according to von Pfundler's research, there is no regular correlation with this condition, any more than there is with lymphatic diathesis. The inflammability of the skin can therefore evidently depend on quite different hereditary factors, which only partly determine susceptibilities in other organ systems at the same time. Exudative diathesis is observed two to three times as frequently in male children as in female children. According to von Pfundler, inheritance is more frequently through the mother than through the father. It can therefore be assumed that some of the hereditary factors involved here are sex-linked recessive traits.

Some researchers associate exudative diathesis with so-called vagotonia, a common and clinically significant abnormality of the autonomic (involuntary) nervous system. The manifestations of vagotonia are varied and changeable: excessive gastric acid secretion, spastic constipation, a globus sensation in the throat, asthma, a tendency to sweat, bradycardia, and so on. External causes play a significant role in triggering the individual symptoms; the anomaly itself, however, is likely to be idiopathic in nature. Bronchial asthma manifests itself in more or less frequent attacks of severe breathlessness, which tend to occur particularly at night. The mucous membrane of the airways is swollen and secretes thick mucus. Asthmatics generally also display neurotic (or, more precisely, psychopathic) symptoms; however, it is not correct to regard asthma simply as a manifestation of hysteria. The attacks can be triggered by physical exertion, colds, emotional excitement and the like. The predisposition to asthma probably does not constitute a strictly defined entity in idiotypical terms. Since true asthma occurs overwhelmingly in men and since transmission via healthy daughters to male grandsons has been reported, one must consider sex-linked recessive inheritance. The exact mechanism is not yet known.

In families with a history of asthma, so-called 'colica mucosa' is more common; this involves the paroxysmal expulsion of strands or even whole tubes of thick mucus accompanied by colic-like pain. Asthmatics also generally have abnormally high numbers of a certain type of white blood cell, known as eosinophils; however, it does not seem appropriate to postulate a specific 'eosinophilic diathesis'. The most appropriate term for the underlying anomaly is likely to be vagotonia.

Similarly, the susceptibility of young children to rickets—which is also, somewhat inaccurately, referred to as the 'English disease'—is largely due to a hereditary predisposition, so that one may well speak of a 'rickets diathesis'. Rickets is accompanied by defective bone formation; furthermore, even normally formed bone can become decalcified and thus soft and pliable. This often results in severe deformities of the bones of the legs, the pelvis and
etc. Tooth development is also impaired. Whilst the primary cause of rickets lies in external factors—such as an inadequate diet, lack of sunlight, etc.—there are families in which, even under favourable conditions, several or all the children develop severe rickets. There is no doubt that there are pathological hereditary factors which predispose individuals to rickets.

Alongside rickets, but often without it, so-called spasmophilia occurs. This includes the majority of so-called
'convulsions', which laypeople are particularly fond of attributing to 'teething'. The most important cause of spasmophilia, however, lies in hereditary predisposition; one may therefore also speak of a spasmophilic diathesis. In some families, the majority of children die from spasmophilic convulsions.

Whether tetany—a neurological disorder accompanied by convulsions, which can be regarded as spasmophilia in adults and which is caused by a failure of the parathyroid glands—is also partly idiopathic has not yet been determined. In any case, the condition has been observed repeatedly in some families.

Another constitutional anomaly, the causes of which are unclear, is so-called Mongolism or Mongoloid idiocy. This involves children with severe mental retardation, whose physical appearance, in a sense, constitutes a caricature of the Mongoloid type. One gets the impression that pathological Mongolism has, for the most part, not been clearly distinguished from the genuine Mongoloid type, which is, after all, widespread in Europe. When this type coincided with idiocy arising for whatever reason, the term 'Mongolism' was often readily applied. The first person to describe the condition, Langdon Down, assumed a connection between the two phenomena. The cause of Mongolism is usually attributed to states of exhaustion in the mother. However, such conditions generally do not affect the foetus in this way at all. One must view these claims with considerable scepticism. The extent to which hereditary factors play a part has not been clarified.

Externally similar to Mongolism is myxedema in children, or so-called sporadic cretinism; it differs from Mongolism in its characteristically doughy yet dry skin, as well as in the fact that it responds favourably to the administration of animal thyroid extract. It arises, in fact, as a result of a failure of

the thyroid gland. The thyroid gland may either be absent from birth or fail during the early years of childhood; in both cases, this is evidently due to idiopathic causes. The severity of the consequences varies depending on the age at which the thyroid failure sets in. Children suffering from myxedema remain dwarf-like in stature and mentally deficient, so-called 'cretins'.

This so-called sporadic cretinism, which is not confined to specific regions, must be distinguished from endemic cretinism, which occurs in areas where goitre is prevalent, particularly among children of mothers with goitre, and which does not appear to be of idiopathic nature but, like goitre in general, seems to be caused by some external harmful factors of an infectious or toxic nature. It is a common occurrence in the Alpine countries, where in some areas almost all women suffer from goitre, and is also found in the low mountain ranges.

In contrast to myxoedema, Graves' disease is characterised by increased thyroid activity. Whilst metabolism is slowed down in myxoedema, the opposite is true in this condition. The thyroid gland is usually also enlarged externally; at the height of the illness, the eyeballs may protrude markedly ('exophthalmic disease'). Graves' disease itself is an acute condition that can set in quite suddenly and then disappear almost completely, though in other cases it can, of course, lead to death within a short period of time. The predisposition appears to lie in a hereditary diathesis. In many people, however, the Graves' diathesis does not lead to the pronounced clinical picture of Graves' disease; instead, the course is more chronic, dragging on for years and decades. The most consistent sign is a persistent rapid heartbeat, followed by a fine tremor of the fingers. A person with Graves' disease resembles someone who is out of breath after running (palpitations, increased metabolic rate, warm, clammy skin, tremors, emotional agitation). Their physical capacity is correspondingly low. As a result of constant overexertion of the heart, cardiac enlargement occurs and, ultimately, heart failure often sets in.

Graves' diathesis is essentially, and certainly, idiopathic in nature. The precise pattern of inheritance, however, has not yet been clarified. There is some evidence to suggest that the genetic predisposition generally manifests as a dominant trait.

Full-blown Graves' disease is found several times more frequently in women than in men. This does not, however, necessarily mean that the genetic predisposition is distributed unevenly between the sexes. It is sufficient to assume that the predisposition manifests itself more frequently and more severely in women, in a similar way to the non-idiotypic goitre is found much more frequently and is more pronounced in women than in men

Fig. 47.
Family with Basedow's diathesis.

. The less pronounced forms of Graves' diathesis are by no means rare in men either. If, for example, it were to transpire that the predisposition to Graves' disease occurs approximately twice as frequently in women, one would have to consider sex-linked dominant inheritance.

A goitre-like enlargement of the thyroid gland also occurs hereditarily in some families, without any other signs of Graves' disease being present. This sporadic goitre appears to be dominantly inherited, possibly sex-linked dominant.

Among constitutional abnormalities, dwarfism should also be discussed here. The term 'dwarfism' or 'nanism' encompasses several conditions, some of which are directly hereditary, whilst others are caused by disorders of the endocrine glands and are thus largely indirectly hereditary. Chondrodystrophy, which is also usually classified as dwarfism, has already been discussed under the section on malformations (see p. 186).

Primordial nanossomia is certainly of an idiotypic nature, manifesting itself at birth in the abnormal smallness of the entire body. In adulthood, these dwarfs exhibit the same body proportions as normal people, with the sole difference that they are simply much smaller.

A second form of idiotypic dwarfism is nanosomia infantilis, in which the skull—and indeed the entire skeleton—retains childlike features well into adulthood. The cartilaginous joints of the bones do not ossify, and the reproductive organs also remain at a childlike stage. It is therefore only to this type of dwarfism that the term ateleiosis applies, which signifies that the normal goal of development is not attained. Nanosomia infantilis can be regarded as an extreme form of infantilism.

In genetic research, these two types of dwarfism have not yet been sufficiently distinguished from one another. The pattern of inheritance has therefore not yet been clarified. From the fact that the parents of dwarfs often have normal stature, whilst there are often other dwarfs amongst their siblings, one may reasonably conclude that there are recessive predispositions to dwarfism. On the other hand, a case has been reported in which 14 children of normal height were born to two parents of dwarf stature. In this case, one must presumably assume that the dwarfism in both parents

was not due to the same recessive genetic factor, but to two different ones, and that in the children each of these factors was masked by the corresponding normal allele inherited from the other parent.

Dwarfism in the form of nanosomia primordialis is not fundamentally different from the racial dwarfism found among the Pygmies of Africa and the islands of India. However, in those cases, dwarfism is not to be regarded as pathological, as it likely represents a selective adaptation to harsh living conditions.

Gigantism, too, is essentially of idiotypic origin. It was already explained in the chapter on normal racial traits that a person's height depends on several genetic factors. Among the endocrine glands, the pituitary gland in particular exerts a significant influence on height. Excessive activity of this organ gives rise to a peculiar form of gigantism, which primarily affects the extremities such as the hands, feet, nose and chin—a condition known as acromegaly. Through experiments with so-called 'Russian' rabbits, which exhibit a hereditary black pigmentation of the extremities, that is to say, a form of 'acromelania', that the 'distal' parts occupy a special position due to their cooler temperature, and that the function of the pituitary gland likely consists in regulating the blood supply and growth of these cooler body parts, which would otherwise be at a disadvantage compared to the rest of the body. Occasional reports have been made of a familial clustering of cases of acromegalic gigantism. This is likely to be a hereditary hyperfunction of the pituitary gland. It is also probable that there are hereditary forms of pituitary insufficiency leading to stunted growth.

The thyroid gland also has a strong influence on growth. In myxoedema, which is caused by thyroid deficiency, children remain dwarf-like in stature. People with Graves' disease, on the other hand, are on average taller than the general average. In regions where goitre is prevalent, cretinism is the most common cause of dwarfism. A characteristic feature here is the indentation of the bridge of the nose, which is a manifestation of growth retardation at the base of the skull and which is found in all cases of generalised growth retardation of the cartilaginous zones of the bones, for example, in chondrodystrophy. Finally, the development of the gonads also has a significant influence on body height.

Delayed longitudinal growth may also result from premature ossification of the cartilaginous joints, where the longitudinal growth of the bones takes place. This, too, appears to run in some families. The accelerated growth seen in childhood comes to a halt relatively early, and here, too, the base of the skull appears to remain relatively short and the bridge of the nose flat.

Of course, purely paratypical factors such as nutrition during development are also significant for body height. Severe rickets can also be the cause of dwarfism, and in this case, the short stature would be influenced by idiotypical factors only to the extent that severe rickets is often idiotypically co-determined.

Constitutional hypertension (hypertension) may also be classified within the realm of constitutional anomalies. Perhaps this, too, is a disorder of the endocrine glands, namely excessive secretion by the adrenal glands. In any case, however, this anomaly also runs in families

. The consequences are extremely varied and serious. Very often, over the years, the renal arteries harden as the normal tissue of the arterial wall is replaced by connective tissue (scar tissue). This nephrosclerosis, in turn, leads to a further rise in blood pressure. The kidney tissue undergoes focal necrosis to varying degrees, resulting in a sclerotic shrivelled kidney, which is always a matter of serious concern. Where renal atrophy occurs frequently within families, without any other conditions or toxic exposures (such as syphilis, gout or lead poisoning) being suspected, hereditary hypertension must be considered. The same applies to the familial occurrence of strokes and heart attacks at a relatively early age (the sixth decade), provided that no external harmful factors such as syphilis, alcohol or nicotine can explain the clustering. Strokes (cerebral infarcts) occur when sclerotic arteries in the brain rupture; heart attacks are usually caused by arteriosclerosis of the coronary arteries, which supply the heart with blood.

Arteriosclerosis consists of patchy hardening of the arteries, which eventually progresses to calcification. It is, in a sense, a form of premature wear and tear of the arteries. Although there are few people of advanced age among us who are completely free of it, the condition nevertheless manifests itself at very different ages in different people and also varies according to its predominant location. External factors such as occupational hazards and toxic effects certainly play a part. At the same time, however, individual predisposition is evidently of the utmost importance. For this reason, arteriosclerosis is particularly prevalent in certain families. In some families, susceptibility to arteriosclerosis is more of a general nature; in others, cerebral arteriosclerosis with strokes is the predominant feature; in yet others, it affects the coronary arteries of the heart; and in still others, the renal arteries, depending on whether familial hypertension coincides with specific organ predispositions.

As a rare constitutional anomaly, osteofibrosis ('ostitis') deformans should also be mentioned here; this usually only sets in later in life and consists of premature failure of the skeletal system. The bones become flexible and spongy, thickening unevenly and becoming interlaced with connective tissue. The process is roughly analogous to arteriosclerosis in the blood vessels. Osteofibrosis, which has been observed with increased frequency in several families, is almost certainly hereditary.

A similar degenerative disease also occurs in the joints, known as a r t h r i t i s d e f o r m a n s. Owing to a constitutional predisposition, but with the contribution of external factors (heavy physical labour, cold), the articular cartilage becomes rough and fibrosed; as the joints become thickened and painful, functional capacity is severely impaired. This predisposition is thought to be linked to Dupuytren's contracture (see p. 179).

Amongst constitutional abnormalities, the hereditary bleeding disorder—which goes by the rather awkward name of haemophilia—is perhaps best discussed here. Whilst in healthy people bleeding from minor wounds soon stops as a result of blood clotting, this clotting process fails to occur in haemophiliacs. Blood can seep from minor wounds for days on end, leading to life-threatening blood loss. Having a tooth extracted becomes a life-threatening operation. Even minor strains or movements can cause haematomas to form in joint cavities, rendering the affected limb unusable for a long time. A large proportion of men with haemophilia die from the condition in the first few decades of life. From the fourth decade onwards, the severity of the condition is said to diminish. The cause of the impaired blood clotting ability has not yet been established.

True hereditary haemophilia has so far been observed only in men. Among women from families with haemophilia, severe blood loss does not occur any more frequently than in other women, not even during menstruation or after childbirth. Haemophiliacs invariably inherit their condition from their mother, who herself is unaffected by it. The genetic trait can also be passed down through the female line across two or more generations. Transmission from father to son, on the other hand, has never been observed. The pattern of inheritance for haemophilia is therefore most likely the same as that for red-green colour blindness, i.e. sex-linked recessive. A definitive determination is so difficult because true haemophiliacs so rarely have offspring.

Fig. 48.
Excerpt from a family with haemophilia. After Fischer.

The best-studied family group with haemophilia is the so-called 'Mampel family', which has become something of a household name precisely because of its hereditary condition. Over time, 37 haemophiliacs have been observed within this family group, who have 29 healthy brothers and 52 healthy sisters. Since it must be assumed that in some branches of the family the predisposition was merely by chance not passed on to any of the sons, even though the mother carried the predisposition, the number of healthy sons is likely to be somewhat higher. Of

the haemophiliacs, five had no healthy brothers. If one now assumes that an equal number of healthy sons of mothers who carried the predisposition happened not to have any affected brothers, one arrives at a ratio of 37 affected to 34 healthy, which is consistent with Mendel's prediction within the margin of error associated with small sample sizes.

Apart from the 'Mampel family', a large number of other haemophiliac families have been described, which are particularly widespread in certain regions such as Württemberg and Switzerland. Some American haemophiliac families appear to trace their origins back to European ones. Some of the family trees from earlier times do not fit the assumption of sex-linked recessive inheritance, but all those that have come to light in recent decades do. One must presumably assume that, in earlier times, conditions were often regarded as haemophilia which in reality bore only an outward resemblance to it. The claim that haemophilia families are particularly large and that there are a disproportionate number of boys among the children, as well as the claim that far more affected brothers are born than healthy ones, can be attributed to the use of flawed calculation methods (see pp. 241–245).

In many medical textbooks and handbooks, a schematic representation of the 'Mampel family' has become widespread, which corresponds neither to the actual circumstances within this family circle nor to the theoretical concepts we can form regarding the inheritance of haemophilia. I include the diagram in question here merely as a warning.

Fig. 49.
Alleged family tree of the 'Mampel haemophilia family', which appears in many medical textbooks and handbooks but corresponds neither to the actual circumstances nor to the theory.

The actual family tree of the 'Mampel family' is too extensive for me to reproduce here . In any case, nowhere in it is there even remotely such a concentration of the disease as in the third generation of the incorrect diagram. Nevertheless, for the sake of comparison, I would like to reproduce at least part of the genuine family tree here.

Fig. 50.
Excerpt from the 'Mampel haemophilia family'. According to L o s s e n.

A condition similar to haemophilia has been described by Glanzmann under the name 'hereditary haemorrhagic thrombasthenia' on the basis of nine family histories. It manifests itself mainly as bleeding from the hands and mucous membranes and is less dangerous than true haemophilia. Nevertheless, almost unstoppable nosebleeds and life-threatening blood loss following tooth extraction do occur. A significant proportion of the cases grouped under the term 'Morbus maculosus Werlhofii' appear to fall into this category. In contrast to haemophilia, not only do female patients occur, but they even seem to significantly outnumber the males. Since the trait is generally dominant, Agnes Bluhm suggests that this may involve sex-linked dominant inheritance.

Von Engelking has described a family in which, over three generations, a number of individuals were afflicted with polycythaemia, i.e. in whom the number of blood cells was excessively high and the total blood volume was also very large. Apart from various subjective symptoms, the condition manifests itself in a bluish discolouration of the skin. Since a similar clinical picture occurs in so-called 'cyanosis', which is caused by a congenital heart defect, it does not seem impossible that hereditary polycythaemia is, at its core, also a hereditary circulatory disorder, and that the increase in blood cells should be interpreted as an imperfect adaptive response.

In chronic familiar jaundice or hereditary jaundice, there is, from birth, a distinctive yellowing of the skin, which is not caused by skin pigment but by bile pigment, which is derived from the blood pigment. The blood thus constantly contains bile pigments. At the same time, the spleen is abnormally enlarged. Evidently, the spleen is constantly destroying blood cells in excessive quantities. The jaundice can be eliminated by surgical removal of the spleen.

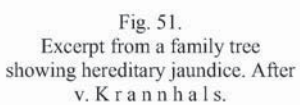

Fig. 51.
Excerpt from a family tree showing hereditary jaundice. After v. Krannhals.

The common form of transient jaundice, which affects many newborns, also appears to occur particularly frequently in certain families , as does the rare, severe form of neonatal jaundice that leads to death.

In the case of hereditary enlargement of the spleen, or splenomegaly, the spleen grows disproportionately in size during adolescence. At the same time, the skin takes on a brownish-yellow colour when exposed to sunlight. The enlargement of the spleen is due to the proliferation of peculiar clusters of cells, which also develop in the liver, the lymph nodes and the bone marrow. Otherwise, this rare condition appears to be harmless. The mode of inheritance is apparently dominant.

It is also worth mentioning here the so-called varicose veins, the development of which is significantly influenced by a sedentary lifestyle or other impairments to venous return, but which do not occur in most people despite such adverse factors. Haemorrhoids represent a specific type of varicose vein; they consist of the dilation of the haemorrhoidal veins around the anus and can be triggered, for example, by a sedentary lifestyle. Like varicose veins in the legs, haemorrhoids tend to run in families.

f) Hereditary metabolic diseases.

The group of metabolic disorders cannot be clearly distinguished from that of constitutional disorders; every metabolic disorder is, in a sense, a constitutional disorder, and vice versa. In particular, all hereditary diatheses also manifest themselves in metabolic abnormalities. In a number of diseases, however, metabolic disorders are so prominent in the clinical picture that it is appropriate to group them together as a separate category.

A typical metabolic disorder is diabetes mellitus. The most prominent symptom is the excretion of glucose in the urine. The body of a diabetic has partially lost the ability to utilise sugar and other carbohydrates for its own maintenance, in particular the ability to store carbohydrates within the body. In addition to the weakness resulting from the body's inability to utilise food properly, various other complications of this metabolic disorder also pose a threat to the life of a diabetic, e.g. metabolic acidosis.

Diabetes usually develops in middle age, and more often still in later life. Most cases appear to be due to a failure of certain groups of cells in the pancreas (the so-called islets of Langerhans), that is, to a disorder of internal secretion. However, as many organs are involved in carbohydrate metabolism, there are probably other causes as well, and consequently other types of diabetes. In many cases, the failure of the islets of Langerhans is undoubtedly due to a genetic predisposition affecting these cell groups. In other cases, however, external harmful factors appear to play a decisive role, in particular syphilis and chronic poisoning. The fact that diabetes is more than twice as common in men is probably not due to sex-linked inheritance, but rather to the fact that men are more exposed to external harmful factors. This is likely why, up until the end of the third decade of life, the incidence of diabetes is just as high among women as

men. In young people, diabetes rarely arises from external causes, and in families with a history of diabetes, we therefore find predominantly juvenile diabetes. From the fourth decade onwards, external influences take on greater significance, and this may explain the predominance of men among those with diabetes in later decades. Since diabetes in women is relatively more frequently idiopathic than in men, this also explains why women pass on the predisposition more frequently than men. This probably has nothing to do with sex-linked inheritance. It is likely that external harmful factors, such as metabolic overload due to intemperance, can at least accelerate the manifestation of a hereditary predisposition to diabetes. Diabetes can, in general, be regarded as premature ageing of certain parts of organs or organ functions, comparable to the premature greying of hair in some families. Different organs age at different rates in different people; and excessive strain accelerates the age-related deterioration of every organ. In some families, diabetes appears to follow a dominant pattern of inheritance; yet it is not unlikely that there are also pathological genetic predispositions with other patterns of inheritance that can cause diabetes.

Fig. 52.
Excerpt from a family tree showing diabetes.
According to v. Noorden.

Of course, as far as the inheritance of the predisposition is concerned, whether or not the condition has already manifested itself at the time of conception. Heredity does not consist in the transmission of physical characteristics or conditions, but in the passing on of idiotypical predispositions inherited from ancestors. For this reason, the so-called 'anticipation' or 'anteposition' occasionally claimed for diabetes, which is illustrated in an ever-earlier onset of the condition over the course of generations. Since the predisposition can manifest itself at different ages even within the same family, it is likely that, in earlier generations, only those diabetics in whom the condition appeared later will have had children. Diabetic women are, as a rule, infertile, and diabetic men, too, rarely go on to have children due to the sexual weakness often associated with the condition. The parents are therefore subject to a form of selection favouring a late onset of the condition. Conversely, in the youngest generation, one will mainly find only those individuals already suffering from the condition in whom it manifested itself early, whilst those siblings in whom the predisposition manifests itself only later are not counted as ill at the time of examination. Added to this is the fact that diabetes is often diagnosed earlier in the offspring of diabetics than in their parents, simply because it is suspected earlier. This, too, contributes to the appearance of 'anticipation'.

Hereditary diabetes is decidedly more common amongst Jews than amongst the rest of the Central European population. According to Teilhaber, of 1,000 Jews who

1910 in Berlin, 80 – that is, 8 per cent – had diabetes.

The so-called renal glycosuria should not be confused with true diabetes mellitus ( ), in which sugar is also excreted in the urine, but which is relatively harmless and appears to be due to a non-progressive renal abnormality. Renal glycosuria is also hereditary and appears to be dominantly inherited.

Another harmless hereditary abnormality of carbohydrate metabolism is pentosuria, in which pentoses are excreted in the urine, apparently regardless of diet.

Greater significance is attached the so-called diabetes insipidus, which shares with diabetes mellitus only the excretion abnormal large volumes of urine, but not the sugar excretion. There is a relatively harmless form of the condition, also known as hereditary polyuria, which is simply dominantly inherited. Whether and in what way the severe forms of the condition, which are thought to be due to a disorder of the endocrine function of the pituitary gland, are hereditary has not yet been clarified.

Fig. 53.
Excerpt from a family tree showing hereditary polyuria. According to Weil.

Cystinuria, or cystine diathesis, consists of a hereditary disorder of protein metabolism. The normal breakdown of the sulphur-containing component of protein is impeded, and this appears in the urine in the form of a sulphur-containing amino acid, cystine. Cystine stones may form in the bladder and cause symptoms. The condition is not entirely harmless in other respects either. The inheritance pattern is simple dominant.

Fig. 54.
Family tree of a family with cystinuria.
According to Abdershalden.

A disorder of protein breakdown also underlies alkaptonuria is also based on a disturbance in protein breakdown. In this case, the breakdown of the benzene ring in the protein is impeded, and homogentisic acid—a normal intermediate product of protein breakdown that still contains the benzene ring—appears in the urine. Such urine turns dark brown to black on exposure to air. Discolouration and deposits also occur in various organs. The condition appears to be simply recessive.

After diabetes, the most important hereditary metabolic disorder is gout (arthritis uratica). Gout sufferers experience extremely painful attacks of joint inflammation, resulting in deposits of uric acid on the joint cartilage . These uric acid deposits can grow into large nodules and

Fig. 55.
A family with alkaptonuria.
According to Ebstein.

severely impair mobility. As uric acid originates from the nuclei of animal cells, the more animal-based foods containing cell nuclei are consumed, the more frequently and severely the gouty diathesis manifests itself in painful attacks and joint changes. Alcohol abuse and lead poisoning can also contribute to the onset of gout. Gout most commonly first occurs in a person's forties; it is several times more common in men than in women.

The predisposition to gout generally appears to follow a simple dominant pattern of inheritance. In some families, the gouty diathesis can be traced back through many generations; in others, the pattern of inheritance appears to be interrupted, though this may be due to the fact that external influences, particularly diet, play a significant role in triggering the condition. Gout is particularly common amongst the Nordic race (coastal regions of the North Sea and the Baltic Sea).

A predisposition to kidney stones is also associated with uric acid diathesis. Kidney and bladder stones composed of uric acid are more frequently found among members of the same family, and gout also tends to occur in these families.

Oxalate kidney stones are also said to run in families. The same has already been mentioned above with regard to cystine stones.

The third in the group of major metabolic disorders is obesity or, as it is termed in its milder forms, adiposity. It manifests itself in an abnormally large accumulation of fat in the subcutaneous tissue, particularly on the abdomen, the lower back, the thighs and elsewhere. Obese individuals can reach weights of 5–6 centners and above. Naturally, diet plays a major role in the development of obesity; it cannot manifest itself in cases of chronic malnutrition. On the other hand, however, obesity cannot simply be induced by overeating in individuals with a predisposition to thinness. There is therefore neither pure 'overfeeding' obesity nor pure 'idleness' obesity. All types of obesity are accompanied by a disturbance in endocrine function. In some cases of obesity, the activity of the thyroid gland is reduced; this gland regulates the rate of metabolism and has been likened to a bellows that fans the flames of the body's metabolic processes. Many cases of obesity appear to be due to inadequate development of the gonads. A particular type of obesity is caused by impaired function of the pituitary gland.

Moreover, in cases of pronounced obesity, more or less all the internal glands usually appear to be functioning poorly. More than half of obese individuals are descended from obese parents, which suggests that there are dominant genetic factors contributing to obesity; however, given the diversity of the clinical picture, it is reasonable to assume that there are also genetic factors with other modes of inheritance.

The possibility of efficient pig fattening is based on the ability to breed for a predisposition to fat deposition in animals. In this case, the relevant genetic trait is regarded as a 'noble' breed trait. Wild boars cannot easily be fattened.

Of course, constitutional leanness is also hereditary. Nutritional requirements vary considerably among different people despite the same body weight and the same behaviour, and these differences are essentially, and certainly, idiotypically determined. People prone to obesity generally have a low metabolic rate and low nutritional requirements, whilst those prone to leanness have a high one.

Constitutional obesity and constitutional leanness are clearly linked to the differences between the major races. The former is unmistakably more common amongst Mongoloids (Chinese, Turks, Magyars, Jews), the latter amongst the Nordic race and its relatives (Anglo-Saxons, Scandinavians, Spaniards, Semites proper, and the German nobility).

So-called lipomatosis, in which fat appears in the form of numerous rounded lumps, and the so-called Dercum's disease, in which painful fatty growths develop, are also evidently idiotypically determined.

It is often reported that the three major metabolic disorders—diabetes, gout and obesity—occur together within the same families. It was also generally assumed that all three were based on the same genetic predisposition and could, in a sense, be represented in the inheritance pattern. This was often referred to as 'heterogeneous' or 'polymorphic' inheritance. Particularly in families where severe forms of metabolic disorders can be traced through the generations, the pattern of 'homogeneous' inheritance is certainly predominant. On the other hand, it is conceivable that mild predispositions to disease may, depending on how they interact with others, lead to various metabolic disorders. Thus, in the case of a predisposition to obesity, a state of overfeeding—and hence an overload of carbohydrates or nucleic acids—arises very easily, and it is easy to understand that, in this way, mild deficiencies in carbohydrate or uric acid metabolism, which would otherwise have had no pathological consequences, can manifest as diabetes or gout. Such relationships are conceivable in many different ways.

The so-called 'arthritis', which in France has often been regarded as the common basis of diabetes, gout, obesity, arteriosclerosis, shrivelled kidney, asthma, psoriasis, muscular rheumatism and various other ailments, is probably also based on similar connections. Obesity places great strain on the heart and arteries, and the heavy burden on the blood vessels can lead to their premature wear and tear, resulting in arteriosclerosis. Furthermore, abnormal chemical composition of the blood in metabolic disorders may well promote the development of arteriosclerosis. On the other hand, arteriosclerosis can lead to diabetes through damage to the pancreas, and so on. Although, therefore, all those conditions grouped under the term 'arthritis' are likely to be at least

, one must not assume that an ‘arthritic diathesis’ constitutes an idiotypical entity.

g) Other internal diseases.

Metabolic disorders are closely related to kidney disorders, in which the entire metabolism is pathologically altered as a result of abnormal excretion. Families have been identified in which kidney atrophy occurs frequently, without any other diseases or harmful factors being identified as the cause. One must presumably assume that this involves an idiotypical weakness of the renal tissue, which causes premature ageing of the cells.

The significance of idiotypical predispositions in the development of kidney inflammation is also to be regarded as established. For instance, there are families in which kidney inflammation has regularly been observed following scarlet fever, whereas this is otherwise by no means a regular consequence of the disease.

Constitutional albuminuria, i.e. the excretion of protein in the urine without detectable kidney damage, has also been observed with increased frequency in some families. Infectious and other kidney disorders also occur more frequently in the same families, which points to an idiotypical weakness of the organ.

A familial pattern has also been described for constitutional haematuria; this manifests as occasional blood in the urine, attributable to renal haemorrhages.

An idiopathic constitutional abnormality also appears to be present in those cases of paroxysmal haemoglobinuria that are not caused by external harmful factors such as syphilis. The condition manifests itself in the excretion of haemoglobin in the urine and is due to the fact that, in the affected individuals, many blood cells disintegrate following exposure to even very slight cold.

In the case of a not uncommon malformation of the kidneys, known as cystic kidney disease—which consists of the renal tissue being permeated by numerous cavities—there have also been numerous reports of its occurrence within families. As this malformation can only be detected during a post-mortem examination, research into its mode of inheritance naturally encounters great difficulties.

A progressive and incurable form of anaemia, pernicious anaemia, has frequently been observed in several members of the same family. In those cases not caused by external factors, the condition appears to involve an internal failure of the sites of blood formation (bone marrow) – a premature ageing of the blood-producing

organs as a result of a weak idiotypical predisposition. At the same time, the secretion of gastric juice regularly ceases. This 'achylia gastrica' has also frequently been observed in other members of the same families and therefore appears to be idiotypically linked to the predisposition to haematopoietic failure.

Even the most harmless form of anaemia, known as chlorosis or pallor, which is observed exclusively in young girls, occurs with increased frequency in some families. Chlorosis can be easily treated by administering iron supplements. Experience also shows that external circumstances play a part in its development. It is certainly linked to a disturbance in ovarian function.

An idiopathic predisposition also appears to play a role in the development of leukaemia. These fatal blood disorders are caused by the uncontrolled proliferation of white blood cells. Depending on the two main groups of white blood cells—the lobulated-nucleated myeloid and the round-nucleated lymphoid—there are also two types of leukaemia. Familial occurrence has been observed in both myeloid and lymphoid leukaemia. However, external influences are probably of even greater significance, as is the case with malignant tumours, to which leukaemias are closely related.

Various congenital heart defects are certainly inherited; however, as these are not readily apparent, their mode of inheritance remains entirely unknown. The most common and generally harmless heart defect is a hole in the septum between the two atria (persistent foramen ovale). This congenital malformation, which is found in almost one in three cadavers, is most likely of idiopathic origin. A hole in the septum between the two ventricles has been traced through several generations of a family. An abnormal connection between the right side of the heart and the aorta (persistence of the foetal ductus arteriosus) has been observed in several siblings. The abnormal narrowing of the pulmonary artery, which results in severe circulatory impairment ('cyanosis') (see p. 202), is also likely to be of idiopathic origin in most cases. Abnormal steepness and abnormally small size of the heart occur in asthenic or hypoplastic constitutions and are hereditary in association with these. So-called cardiac neuroses are generally thought to be the manifestation of general anomalies of the nervous system, the hereditary nature of which remains to be discussed.

Idiotypical predisposition is certainly also significant in the development of heart defects that follow on from inflammation of the heart valves. Heart valve inflammation (endocarditis) usually occurs in the wake of infectious rheumatic fever (polyarthritis), but is not infrequently observed on its own. Rheumatic fever, heart valve inflammation and, consequently, heart defects are very often observed in clusters within families.

Fig. 56.
Family tree of a family with heart defects following endocarditis. After Strebel (abridged).
It is doubtful whether the woman marked with a question mark
suffered from heart disease. The individuals marked in black also suffered from lens displacement.

With regard to the family described here, one must consider sex-linked dominant inheritance. Furthermore, a predisposition to endocarditis certainly does not constitute an idiotypical entity. It was mentioned earlier that it is often associated with a predisposition to erythema exsudativum. In some families, so many members develop valvular endocarditis that, even assuming dominant inheritance, the predisposition seems to lead to the disease in almost every case. It appears that the opportunity for a rheumatic infection arises from time to time for all those who are susceptible to it, whilst those who are not susceptible do not seem to develop the disease despite repeated opportunities for infection.

Heart defects also occur, predominantly in women, which are not preceded by valvular endocarditis or rheumatic arthritis; these are known as mitral stenosis (narrowing of the opening from the left atrium to the ventricle). These appear to be congenital defects; and since mitral stenosis is said to be about twice as common in women as in men, it cannot be ruled out that most cases of mitral stenosis may be due to a sex-linked dominant predisposition. Moreover, as congenital heart defects are predominantly found in male children, it stands to reason that some of these may follow a sex-linked recessive pattern of inheritance. In any case, the sex chromosome appears to play a particular role in the constitution of the heart.

Idiotypic predisposition is also of great significance in the development of stomach ailments, perhaps even more so than an inappropriate and excessive diet. 'Dietary errors' lead to more serious health problems almost exclusively in individuals with
a 'weak stomach' to more serious health problems. Muscle weakness and flaccidity (atony) of the stomach are found particularly in those with an asthenic constitution. Similarly, insufficient secretion of gastric juice (achylia) is largely idiopathic in nature. 'Nervous stomach ailments' occur as a secondary symptom of general anomalies of the nervous system.

Excessive secretion of hydrochloric acid (hyperchlorhydria) is more common in some families, usually as a manifestation of vagotonia (see p. 192). This also includes the spasmodic closure of the pylorus (pylorospasm), which manifests itself mainly in childhood as recurrent, agonising vomiting. Hyperchlorhydria promotes the development of so-called gastric ulcers, which result from the self-digestion of sections of the gastric wall and, accompanied by severe bleeding, can lead to prolonged bed rest and even death. A familial clustering of gastric ulcers has frequently been observed.

In the case of chronic constipation (habitual constipation), the idiotypical predisposition is likely to be just as significant as it is for the (rarer) tendency towards diarrhoea, from which some individuals suffer throughout their lives. Spastic (cramp-like) constipation is usually a secondary symptom of vagotonia, whilst atonic (flaccid) constipation is associated with asthenia. So-called Hirschsprung's disease, in which an abnormally wide large intestine leads to the accumulation of large masses of faeces in the large intestine even in early childhood, is probably also idiotypically determined.

Liver atrophy (liver cirrhosis) does indeed tend to develop under the influence of toxic effects (alcohol, syphilis, malaria); but since these harmful factors affect countless people without leading to liver cirrhosis, one must surely consider the possibility of an idiotypical organ weakness; indeed, the condition has frequently been observed to run in families. Premature wear and tear due to constitutional weakness is, after all, known to occur in several organs.

Nothing definite is known about the hereditary nature of gallstone disorders. Since external factors (e.g. a pressure mark caused by ill-fitting women's clothing) play a significant role in their development, and since gallstones do not cause any particular symptoms in many of those affected, the question of a hereditary predisposition to them is very difficult to investigate.

As regards diseases of the respiratory tract and the lungs, chronic bronchial catarrh—and, more frequently associated with it, emphysema—is not infrequently found to run in families. The aforementioned predisposition to asthma can also lead to these secondary conditions. On the other hand, an idiopathic 'rigid dilatation of the thorax'—in which the chest remains fixed in a position as if at the peak of inhalation—also appears to be a potential cause of chronic emphysema and bronchial catarrh.

By far the most significant lung condition is pulmonary tuberculosis, which was long regarded in the past as a hereditary constitutional disease and then, following the discovery of the tuberculosis bacillus, as a purely paratypically caused disease. The truth is likely to lie somewhere in between. Without infection with the pathogen, tuberculosis naturally does not develop; yet, under present-day living conditions, the vast majority of people become infected with tuberculosis as early as childhood, and yet only a certain fraction of them later develop pulmonary tuberculosis. In any case, the marked familial clustering of tuberculosis cannot be explained solely by transmission within the family. Nor, on the other hand, is it certainly due solely to a shared hereditary susceptibility. External factors such as malnutrition and occupational hazards also play a significant role.

And yet pulmonary tuberculosis is a disease with a hereditary component. If the parents or siblings of a patient have died of consumption, experience shows that their prospects of recovery are particularly poor. In other families, the course of the disease is consistently milder. Whilst hereditary differences in predisposition may not play a role in the tuberculosis infection itself, they certainly do so all the more in the development of consumption.

Hereditary susceptibility to tuberculosis is evidently not uniform in nature. Only a few of the pathological predispositions that may be responsible for this are known with any degree of certainty. There can be no doubt that an asthenic constitution (see p. 189) entails an increased susceptibility to pulmonary tuberculosis. The experience of life insurance companies has shown that individuals whose chest circumference is too small in relation to their height are affected by consumption in an unusually high proportion of cases. Opponents of a hereditary predisposition to tuberculosis have, admittedly, attempted to portray the asthenic constitution as merely a consequence of a tuberculous infection in childhood; however, this is contradicted by the fact that asthenia is distinctly hereditary; moreover, there are many asthenic individuals who never develop tuberculosis. The significance of malnutrition in the development of consumption is generally acknowledged. It follows, however, that all those hereditary predispositions which lead to nutritional disorders and anaemia also indirectly cause a susceptibility

to tuberculosis. In diabetes, for example, consumption often develops because blood containing sugar provides a particularly favourable breeding ground for the bacilli. Thus, the hereditary predisposition to diabetes is at the same time a predisposition to tuberculosis. There is therefore not just one hereditary predisposition to tuberculosis, but many.

Until recently, it was often thought that the question of the 'hereditary nature' of tuberculosis could be dismissed by pointing out that hereditary transmission from the mother to the foetus in the womb generally does not occur, and that such transmission from the father's sperm is certainly impossible. This, of course, is based on a complete misunderstanding of the nature of heredity. It is also linked to a lack of understanding of heredity when people assume that, in the case of a direct hereditary predisposition, the predisposition would be equally likely to be present in all the children of a couple. Even if a pathological genetic predisposition that entails a particular susceptibility to tuberculosis is dominant, on average it will still not manifest itself in half of the children; with a small number of children, therefore, it will often not manifest at all. A recessive genetic predisposition, on the other hand, will generally not manifest itself in the parents. Such findings therefore do not contradict the hereditary nature of predispositions to tuberculosis. The fact that the children of parents with consumption largely develop the disease themselves does not, of course, in itself prove the hereditary nature of the predisposition. Rather, it is precisely the fact that quite a few children of consumptive parents never develop consumption themselves, despite every opportunity for infection and despite all the adverse circumstances of their lives, which provides much stronger evidence for the significance of heredity.

h) Hereditary neurological diseases.

The term 'nervous disorders' is used here to refer only to those diseases of the nervous system which manifest themselves predominantly through physical symptoms (e.g. paralysis). Those disorders of the central nervous system which manifest themselves predominantly through psychological abnormalities are to be discussed as such, even if, like neurasthenia and hysteria, they are euphemistically referred to as 'nervous disorders'. Some nervous disorders, such as optic atrophy, which manifest themselves in very specific organs, or, like Graves' disease, where the disturbance of the nervous system does not constitute the actual underlying cause, have already been discussed above.

Of particular significance is a group of severe, progressive nervous disorders which are based on the premature degeneration of parts of those nerve pathways that lead from the brain to the voluntary muscles and transmit the impulses for movement to the muscles. These include, first and foremost, various types of hereditary muscle wasting (muscle atrophy).

Hereditary muscle wasting most commonly presents as progressive pseudo-hypertrophic muscular dystrophy. Certain muscle groups gradually weaken and become increasingly paralysed, although externally they may even appear to increase in size due to the simultaneous proliferation of fatty tissue. Patients eventually become completely helpless, until they succumb to the condition after years or decades. Although the nerves appear intact on the surface, the peculiar nature of the progression leads one to conclude that the underlying condition is not a muscular disorder but, in fact, a neurological one. It involves a failure of that nerve function which regulates the proper nutritional status of the associated muscles. There are certainly a whole range of idiotypically different types of muscle atrophy. In some families, the condition begins as early as childhood; in others, during adolescence; and in yet others, only later in life. Furthermore, in different families the condition begins in different muscle groups, for example in the muscles of the pelvic girdle, the shoulder girdle or the face. Some of these different forms appear to represent transitional stages between others, so that it has been suggested that they are merely different manifestations of one and the same predisposition. However, this is strongly contradicted by the fact that, within the same family, the condition regularly

tends to follow a very similar course, and, above all, that the pattern of inheritance can vary considerably between different families. Simply dominant forms are relatively rare; indeed, these generally present a relatively mild course; the severe, rapidly progressive forms would, after all, be unable to persist at all if inheritance were dominant. Similarly, in the family tree described by K e h r e r—which shows dominant inheritance and is illustrated here—the condition took an exceptionally mild course.

Fig. 57.
A family with progressive muscular dystrophy (progressive muscle wasting) according to Kehrer.

In the above family tree, the simple dominant pattern of inheritance is particularly striking in that an affected man, in succession, fathered both healthy and affected children with two healthy women.

Recessive forms are more common, as is evident, for example, from a comprehensive study by Prof. W. Weitz in Tübingen, who has shed light on the heredity of muscular dystrophies through a modern approach. This category presumably also includes many of those cases which are observed only sporadically within a family; out of 71 observations, 54 were 'isolated', 12 'familial' and only one involved 'direct heredity', with multiple cases within the same family being counted as a single observation. If most predispositions to muscular dystrophy are recessive, then such an actual distribution is entirely consistent with theoretical expectations.

Fig. 58.
A family in which two first cousins produced children who later developed muscular dystrophy. According to Weitz.

Sex-linked recessive inheritance also appears to occur with some frequency; whilst in most families men and women are affected in roughly equal numbers, in these cases only men are regularly affected.

Less common than pseudohypertrophic muscular dystrophy is so-called neural progressive muscular atrophy, in which the loss of nerve function is also manifested by recognisable atrophy of the affected nerves. Muscle wasting tends to manifest itself initially in the muscles of the feet supplied by the peroneal nerve; later, the muscles of the hands, arms and legs are also

are also affected. Generally speaking, about five times as many men as women are affected. The predisposition is apparently, at least in most cases, sex-linked recessive.

Fig. 59.
Excerpt from a family tree showing p r o g r e s s i v e m u s c l e a t r o p h y (progressive muscle wasting). After H e r r i n g h a m.

Related to neural muscular atrophy is spinal muscular atrophy, in which those groups of ganglion cells that form the so-called anterior horns of the spinal cord degenerate, leading to the subsequent degeneration of the nerve fibres (neurites) emanating from them. Spinal muscular atrophy usually does not begin until the fourth decade of life, starting with wasting of the hand muscles. In the families of those affected, no further cases are usually detectable; nevertheless, the condition has been observed more frequently in siblings. This points to recessive inheritance. The form of spinal muscular atrophy that begins in infancy and is more frequently observed in several children within the same family is certainly recessive.

The so-called bulbar paralysis also belongs to this group; it consists of wasting of the muscles of the tongue, mouth, pharynx, larynx and oesophagus, and is caused by the degeneration of ganglion cells in the nuclei of the medulla oblongata. As neither speaking nor swallowing is soon possible, the condition usually lasts only a few years. Bulbar paralysis generally occurs as an isolated case; however, cases have occasionally been observed among siblings. It may therefore be reasonably assumed, by analogy with other forms of muscular atrophy, that it also arises on an idiotypic basis; specifically, recessive inheritance is a possibility.

The entire group of muscular atrophies can, in a sense, be regarded as premature ageing of certain parts of the body, comparable to the premature greying of hair in some families and also to numerous other hereditary conditions such as diabetes, cataracts in middle age, primary (idiopathic) renal atrophy, and so on.

Hereditary spastic spinal paralysis (spinal cord paralysis) is caused by the degeneration of that part of the nerve pathway which runs through the spinal cord (pyramidal-lateral tract pathways). Usually beginning in the third decade of life, a distinctive stiff (spastic) paralysis of the legs occurs, such that independent walking is no longer possible. The prognosis is just as poor as for the conditions discussed previously; von Pfundler reported on a family in which all six children of a consanguineous couple developed spastic spinal paralysis as early as infancy.

It is stated that the clinical picture of spastic spinal paralysis can also be brought about by external causes. However, one must bear in mind that even isolated cases may well be hereditary in origin, a fact which has largely been overlooked to date.

In so-called a m y o t r o p h i c l a t e r a l s c l e r o s i s, which is similar to the condition just discussed, ganglion cells of the anterior horns are also destroyed, and consequently, in addition to spastic paralysis, muscle atrophy is also present. It would therefore be more appropriate to describe the condition as spastic muscular atrophy. Familial occurrence has been observed on several occasions; external causes, however, have not been identified.

Nor can it yet be ruled out with certainty whether pathological genetic factors might play a role in so-called Little's disease, a congenital condition in which children fail to learn to walk properly as a result of spastic stiffness in the legs. In most cases, however, the condition appears to arise from damage to the brain during birth.

Progressive spastic paraplegia (diplegia, bilateral paralysis), which is similar to Little's disease, is, however, definitely hereditary. The condition, which is accompanied by nystagmus, strabismus, impaired vision and progressive mental impairment, appears to be recessive. Several forms have been described. The onset of the condition also varies between families; it has been observed in infancy, childhood and even in later adolescence.

If such a hereditary condition were to begin before birth, it would probably be described as Little's disease; therefore, the possibility cannot be ruled out that some of the conditions grouped under this name may also be hereditary.

The group of idiopathic spastic paralysis conditions also includes a hereditary disease described by Pelizaeus and later studied in greater detail by Merzbacher; however, to my knowledge, it has so far only been observed within a single family. In this condition, the white matter of the brain—which, in contrast to the grey matter of the cerebral cortex containing ganglion cells, consists almost entirely of nerve tracts—is severely underdeveloped , a condition resulting from a failure of the so-called myelin sheaths of the nerve fibres. The condition manifests itself shortly after birth and usually leads to death towards the end of the second decade of life. Although the condition is of no practical significance due to its rarity, it is nevertheless of great theoretical interest because its sex-linked inheritance demonstrates that certain genetic factors located on the sex chromosome play a decisive role in the development of the brain.

Fig. 60.
Excerpt from the family tree described by Pelizaeus. After Merzbacher.

Since two affected females are also recorded in a series of siblings, one would have to assume, according to the theory of sex-linked recessive inheritance, that the father of these sisters must also have carried the trait, whereas otherwise the affected individuals would not have reproduced. Unfortunately, nothing is known about the father.

Fig. 61.
A family with hereditary spinal ataxia.
According to Hübscher.

Whereas in the case of muscle atrophies the basis of the disease lies in the wasting away of parts those nerve pathways leading from the brain to the muscles, there are also hereditary neurological disorders in which parts of the pathways leading to the brain are destroyed. These include, above all, hereditary spinal ataxia (spinal ataxia) or Friedreich's ataxia. The condition begins in the first or second decade of life with movement disorders; when standing, sufferers stagger, and with their eyes closed they are completely unable to maintain an upright posture, because the sensory input that constantly provides unconscious control of the limbs fails. Hereditary spinal ataxia occurs more frequently in several children of healthy parents, particularly those who are blood relatives. It is therefore reasonable to assume that it is inherited in a recessive manner.

Hereditary cerebellar ataxia, which is caused by the degeneration of cerebellar pathways, manifests itself primarily as a wobbly, staggering gait; it usually begins in the third decade of life and only leads to death after many years. In a family described by Classen, cerebellar ataxia did not set in before the sixth decade of life. In this case, as in other families, the trait was simply dominant.

Fig. 62.
Family tree of a family with cerebellar ataxia. After C l a s s e n.

Hereditary spinal ataxia is similar to that seen in syphilis, which is known as tabes dorsalis. As only a small proportion of syphilitics develop this condition (1–3 per cent), it is possible that an idiopathic predisposition plays a role. This is not known for certain.

It also remains unclear whether a hereditary predisposition plays a role in the development of syringomyelia, a fatal disease caused by the formation of cavities in the spinal cord.

On the other hand, it is fairly certain that the (rare) condition known as pseudo-sclerosis or diffuse sclerosis is idiopathic in nature; this is but is distinguished by the absence of detectable lesions in the spinal cord. As it has been observed more frequently in two or three children of healthy parents, it appears to be a recessive condition.

Paralysis agitans, or shaking palsy, is a not uncommon condition that usually begins in the fifth or sixth decade of life and manifests itself in peculiar, repetitive, involuntary movements that cease only during sleep. The hands, in particular, are constantly in motion, as if counting coins. Furthermore, a peculiar muscular stiffness develops, giving the face a mask-like expression and forcing the patients into a stooped posture. Lundborg found that in 6 out of 7 cases, the parents were blood relatives. Accordingly, recessive inheritance is likely to be the cause. Cases that manifested at a younger age have occasionally been observed more than once within the same family. By contrast, those predisposed to the common form—which only sets in later in life— —often do not live to experience its onset.

Hereditary chorea (Huntington's disease) is characterised by involuntary movements of a completely different nature. The condition begins in the fourth or fifth decade of life with twitching, often peculiarly theatrical movements of various parts of the body. As the incurable condition progresses, progressive mental deterioration also sets in. The mode of inheritance is simple dominant. Davenport reported on 962 cases from four families. The clinical picture varied somewhat across the different families. Hereditary chorea therefore does not appear to constitute a strictly idiotypical entity either. However, no exceptions to the dominant mode of inheritance were found.

Hereditary chorea is similar to tic disorder, which manifests itself even in young people in the form of distorted movements. Idiotypic causation is not unlikely; yet nothing certain is known. Nor is it known whether hereditary factors play a part in the development of transient chorea (St. Vitus' dance), which often follows infectious diseases such as rheumatoid arthritis.

Progressive hereditary myoclonus, or myoclonus epilepsia, is also based on involuntary muscle twitches; however, the individual twitches are so brief that they do not result in larger movements. At the same time, nocturnal epileptic seizures occur, and ultimately this leads to mental retardation. Lundborg observed 17 cases in children of consanguineous parents. The numerical ratio he found corresponds very closely to recessive inheritance, so that this can be regarded as established.

Hereditary myotonia (Thomsen's disease) is a condition in which all muscles, when tensed after a prolonged period of rest, enter a state of tense rigidity that only gradually subsides. The inheritance pattern is simple dominant.

If the muscle rigidity occurs only in the cold, the condition is referred to as p a r a m y o t o n i a. This has been observed across six generations of a family and is therefore evidently also simply dominant.

The same applies to paroxysmal paralysis or myoplegia, which manifests itself in peculiar episodes of flaccid paralysis affecting almost the entire voluntary musculature.

Fleischer has described a peculiar hereditary condition as 'myotonic dystrophy', which he was able to trace within a large family circle and in which, amidst spasms in some muscle groups and atrophy in others, the affected individuals ultimately succumb to general deterioration. An early sign of the disease is clouding of the lens. The pattern of inheritance has not yet been fully established.

Hereditary tremor (so-called 'Tatterich') can manifest itself even in young people. It occurs as a secondary symptom of other conditions, such as Graves' disease, but also as a result of a specific dominant predisposition.

That peculiar eye tremor, which is known as nystagmus and which has been mentioned, for example, in connection with night blindness and ocular albinism, also occurs without any other eye disorders in conjunction with involuntary head movements, and appears to be the predominant symptom.

Fig. L. 63.
Family tree of a family with hereditary involuntary eye and head movements.
According to Radloff (abridged).

In the chapter on skin disorders, already mentioned several hereditary disorders of the cutaneous vascular nerves, which may also be regarded as neurological disorders. Related to this group is probably the intermittent joint hydrops (hydrops articulorum intermittens), which primarily affects the knees. The question of its hereditary nature has not yet been investigated in detail. The same applies to various nutritional disorders of the skin and other tissues, such as scleroderma and localised facial atrophy (hemiatrophia faciei), in which, without any apparent external cause, the skin, adipose tissue and, to some extent, the musculature of one half of the face atrophy.

So-called bedwetting (nocturnal enuresis) must also be regarded as a manifestation of an anomaly of the nervous system; it is a very troublesome and embarrassing condition characterised by the involuntary emptying of the bladder during sleep. It is caused by a lack of sensation regarding the bladder's state of fullness. In most cases, bedwetting occurs more frequently within certain families and is therefore evidently of idiopathic origin. Bedwetters are relatively often mentally retarded or epileptic; they are also more frequently affected by malformations of the spinal cord (spina bifida occulta).

Left-handedness is also attributable to an anomaly of the central nervous system. Since the right side of the body, which is normally favoured, is controlled by the left hemisphere of the brain, left-handedness suggests a relative functional weakness of the left hemisphere compared to the right. Left-handedness certainly does not constitute a single, distinct condition; rather, this phenomenon may well be caused by various anomalies. This is likely to explain why other disorders resulting from anomalies of the central nervous system

are found with above-average frequency among left-handers or their relatives, e.g. speech disorders, epilepsy and mental retardation. Overall, approximately 3–5 per cent of the Central European population are left-handed; about half of all left-handers also have left-handed relatives. Some of the brain abnormalities on which left-handedness may be based appear to be dominantly inherited, whilst the majority are probably recessive.

The most common speech disorders, stammering and stuttering, are also largely hereditary. In stammering, consonants at the beginning of a word are repeated and prolonged, followed by pauses, so that speech is frequently interrupted and choppy. Stammering refers to the inability to pronounce certain sounds correctly. These conditions are also caused by abnormalities of the central nervous system. Stammering affects around 1 per cent of our population. More than 25 per cent of all people who stammer also have relatives who stammer. The decisive role of hereditary predisposition is particularly evident in cases where children of people who stammer, who were separated from their stammering parent from birth (e.g. due to the parent's death), nevertheless developed the same speech disorder. Furthermore, of several siblings, usually only one or two develop stammering. Among people who stammer, nearly a quarter are left-handed, and among left-handers, the proportion with speech disorders is even higher. Psychopathic conditions are also more common than average among people who stammer and their relatives (epilepsy, hysteria, mental retardation).

So-called 'hear-deafness' – the term used, in contrast to deaf-mutism, to describe the condition of children who, despite having normal hearing, do not begin to speak at the appropriate age – is also usually of idiopathic origin. In more than a third of cases, delayed or incomplete development of speech has also been observed in one of the parents.

Heredity also plays a significant role in lisping (sigmatism); this involves the tongue touching the teeth when pronouncing sibilant sounds. Gutzmann found that in cases of lateral sigmatism (lateral lisping), the same speech defect was present in 38.5 per cent of relatives. A particularly large number of Jews are prone to a peculiar, slight lisp; this appears to be a specific predisposition among certain racial groups within the Jewish population, which, however, can hardly be regarded as abnormal.

i) Hereditary mental illnesses and psychopathies.

In no other group of diseases is heredity so prominent a cause as in mental disorders; at the same time, however, the investigation of the specific patterns of inheritance presents no greater difficulties in any other group. Even regarding the definition and classification of hereditary mental disorders, there has so far been no real consensus amongst experts. It is therefore no wonder that the mode of inheritance has so far been established with complete certainty in almost none of them. The only certainty is that heredity outweighs all other causes in significance.

> In second place is syphilis, which is not only the principal cause of paralysis or so-called 'brain softening', but which, in its congenital form, can also give rise to various forms of mental deficiency and epilepsy. Third place is taken by alcoholism, which can result in delirium tremens and alcoholic dementia in the drinkers themselves, on the one hand, and mental retardation and other mental disorders in their children, on the other. Fourth place is taken by those as yet not fully understood factors which cause endemic goitre and cretinism.
>
> For practical reasons, mental disorders are usually classified into actual mental illnesses (psychoses) and psychopathies, based on the degree to which those affected are able to adapt or function in practical life. An individual is to be regarded as mentally abnormal or disturbed if, as a result of their mental constitution, they live at the limits of their ability to adapt. If the disorders are so severe that the sufferer is unable to pursue a profession, this is referred to as mental illness. In the case of disorders of a lesser degree, which, whilst still compatible with the pursuit of gainful employment, nevertheless entail either a permanent or occasional impairment of the individual's own well-being or of society, the term 'psychopathy' is used.
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> This classification, which is necessitated by practical considerations, corresponds very little to that which research into heredity must aim for. The aim here is to identify the individual pathological hereditary factors in their various possible manifestations and to determine their mode of inheritance. It is to be hoped that, with the aid of research into heredity, it will one day be possible to carry out a genuine classification of mental disorders based on their essential nature, something which, on the basis of clinical research, has so far been achieved only very imperfectly. It is clear from the outset that there will be a whole range of different hereditary mental disorders. Just as with any other organ, there are naturally also numerous hereditary anomalies of the brain, the function of which corresponds to mental processes.

Since an idiotypical classification of mental disorders is not yet possible today, it is inevitable that we must content ourselves here with a classification based on the clinical picture. Essentially, the classification should be

based on Kraepelin's classification, which has probably received the most recognition of all to date.

The terms 'mental deficiency' and 'idiocy' are used to encompass a range of conditions in which normal intellectual development fails to occur from the outset. If the person is capable of pursuing an independent occupation, one does not usually speak of mental deficiency, even in the face of considerable intellectual impairment. Mental deficiency or imbecility, on the other hand, only begins beyond this threshold; yet those with mental deficiency can still be encouraged to engage in all manner of useful activities, whereas this is no longer possible in the case of idiocy or imbecility. Idiots do not even learn to speak. This classification therefore concerns only a difference of degree, and each of these two groups encompasses a wide range of degrees of mental deficiency; naturally, there are also all manner of transitional stages between the two. Mental deficiency can be a symptom of very different pathological conditions, some hereditary and some caused by external factors. One cannot therefore simply speak of the heritability of 'mental deficiency' or 'idiocy'.

A not inconsiderable proportion of cases of mental deficiency is caused by congenital syphilis. According to Kraepelin, at least one third of severe forms of idiocy are attributable to syphilis. Parental alcoholism is thought to play a role that is perhaps just as significant. A further large proportion of mental deficiency is a secondary manifestation of cretinism. However, after excluding all those cases which are caused by external harmful factors, there still remains a large—and, I believe, the larger—proportion of all cases of mental deficiency, which is essentially of an idiotypical nature. This need not contradict the assumption that it is precisely the most severe cases that are usually caused by external factors. The American researcher G o d d a r d, who has specialised in the study of mental deficiency, has concluded that at least two-thirds of all cases of mental deficiency are hereditary. This estimate is consistent with our own experience, since alcoholism plays no role in America and syphilis and cretinism also appear to be less common there than here. In the 'Kallikak family' described by Goddard, 40 marriages between two mentally retarded individuals produced 220 mentally retarded

and only 2 apparently normal children. Marriages between mentally retarded individuals and normal individuals produced mentally retarded children in about half of the cases. Furthermore, mental retardation could be traced in an unbroken line through five generations. This suggests, at any rate, that there are simple dominant genetic factors which cause mental retardation. Davenport and Weeks, on the other hand, have concluded, based on observations of many family groups, that there is a recessive gene for mental deficiency. Since people with mental deficiency very often marry amongst themselves, an unbroken line of inheritance could also be traced through the generations in the case of recessive inheritance. Idiocy, of course, cannot be based on a simple dominant genetic trait, as individuals with idiocy never reproduce; yet it seems possible that certain pathological genetic traits cause mental deficiency when present in a heterozygous state and idiocy when present in a homozygous state. Presumably, there are both dominant and recessive genetic traits that cause mental deficiency.

'Amaurotic idiocy' refers to a severe hereditary disorder that occurs in certain Jewish families and which, in children born apparently normal, manifests itself in the first or second year of life as mental retardation, paralysis of the limbs and blindness. The trait appears to be simply recessive. Similar to amaurotic idiocy are a number of rare hereditary conditions that only develop in later childhood. For instance, there are families in which several children, in the second half of their first decade of life, show a decline in mental faculties and, at the same time, in eyesight, which progresses over the years to the most severe mental retardation (Spielmeyer's disease). Unlike amaurotic idiocy, these other conditions—which also appear to be simply recessive—have not been observed in Jewish families. One must not, of course, regard this as a 'racial predisposition'. Rather, one must assume that this predisposition arose by chance as an idiovariation in a Jewish family and then spread, unrecognised, amongst the Jewish population until, through a chance coincidence of the predisposition in a married couple, a homozygous manifestation became possible.

One of the most common mental illnesses—and, after the paralysis caused by syphilis, arguably the most significant of all—is schizophrenia or dementia praecox, also referred to, somewhat inaccurately, as 'juvenile insanity'. The condition usually sets in during the second or third decade of life, though more often it does not appear until later. It can manifest itself in quite varied forms; what they all have in common, however, is an extensive destruction of emotional and volitional life.

Patients are dominated by senseless impulses and senseless inhibitions. Uncontrolled motor impulses can lead to unbridled rampage; conversely, sufferers may for long periods sink into a state of complete rigidity and paralysis of the will, as in one of the most severe forms, c a t a t o n i a. On the other hand, the condition may also present from the outset as a simple, insidious deterioration of mental faculties: D e m e n t i a

s i m p l e x. Hebephrenia is characterised by a particular disorganisedness of thought. In other cases, hallucinations and disorganised delusions may dominate the clinical picture: D e m e n t i a p a r a n o i d e s. In most cases, schizophrenia progresses in episodes with fairly sudden exacerbations, which may occasionally be interrupted by periods of improvement, until the condition finally comes to a standstill as a result of mental deterioration.

Apart from a hereditary predisposition, no other causes of schizophrenia are currently known. Rüdin, who was the first to tackle the heritability of mental illnesses using a truly modern approach, found, based on data from 701 families, that 4.5 per cent of the siblings of patients also developed schizophrenia when both parents were healthy. If one of the parents was affected, the percentage of affected siblings was 6.2% (34 families). Only 0.6% of step-siblings developed the condition. These findings suggest, to a certain extent, that the predisposition is inherited in a recessive manner. On the other hand, Rüdin found only 14 instances, i.e. 2 per cent, of first-cousin marriages between the parents amongst 700 cases, which tends to argue against recessive inheritance.

Schizophrenic clinical pictures appear to be capable of occurring in connection with quite different underlying conditions. Thus, the picture of catatonia may occasionally occur in individuals who later show no further signs of illness, whilst in most cases mental retardation is the consequence. It must therefore provisionally be regarded as possible that schizophrenic states may be reactions to quite different causes of illness, among which paratypical causes may also be included. Those with a predisposition to schizophrenia usually display milder anomalies, including a blunting of emotional and volitional life, even before the onset of actual mental illness; furthermore, families of schizophrenics generally also include other such 'schizoid' psychopaths, in whom, however, the onset of a pronounced mental illness need not occur and, in most cases, does not occur. It cannot yet be ruled out that there may also be dominant genetic predispositions to schizoid psychopathy and that, on this basis, episodes of severe schizophrenic mental illness may occasionally be triggered by some as yet unknown causes. On the other hand, it is also possible that schizoid psychopathies are merely sequelae of milder episodes of destructive processes.

Given everything we know about other hereditary diseases, such as muscular atrophies, it is highly unlikely that the group of schizophrenias constitutes an idiotypical entity. Nor is it likely that the various clinical presentations can be explained solely by the varying interaction of several genetic units, each of which determines a specific group of symptoms. The characteristic relapsing course and the fact that these relapses are underpinned by destructive processes in the cerebral cortex suggest, rather, a comparison with conditions such as optic nerve atrophy or glaucoma

, which also occur in both more insidious and more acute forms and which also tend to affect certain age groups. Unfortunately, all we know so far is that heredity is a major cause of schizophrenia.

The term 'epilepsy' encompasses conditions characterised by abnormal susceptibility of the cerebral cortex, which usually manifest as episodes of loss of consciousness accompanied by distinctive convulsions. A significant proportion of all cases of epilepsy are triggered by alcohol abuse; however, as most drinkers do not develop epilepsy, hereditary factors are evidently also significant in 'alcoholic epilepsy'. The clinical picture of epilepsy can also develop as a result of purely parakinetics causes, such as brain injuries or infectious brain diseases in childhood. The term 'genuine epilepsy' is used to describe those forms in which no external triggers can be identified. In severe cases, the condition usually leads to mental retardation, which may be anatomically manifested by certain areas of damage in the cerebral cortex. Epilepsy may also present itself under the guise of other mental disorders, for example in episodes of uncontrollable restlessness or in states of clouded consciousness without convulsions (so-called twilight states).

Around 10 per cent of all people with epilepsy have a parent with epilepsy. Since Davenport and Weeks almost invariably found mental deficiency in the families of people with epilepsy, and since people with epilepsy themselves are often mentally deficient, one may reasonably conclude that there are certain hereditary factors which can manifest themselves, on the one hand, as mental deficiency and, on the other, as states of cerebral cortex excitation that cause epileptic seizures. One can envisage the connection in a similar way to that mentioned in relation to left-handedness. Steiner found that around 20 per cent of epileptics were left-handed, and in a further 70 per cent of cases there was a history of left-handedness in the family. This suggests that, in many cases, epilepsy is based on an abnormal susceptibility specifically of the left hemisphere of the brain, which, after all, controls the right hand. The decisive importance of heredity is strikingly demonstrated by a case in which epilepsy manifested itself in completely identical seizures in two identical twins. Davport and Weeks assume that epilepsy is inherited in a recessive manner. However, there are probably also genetic predispositions involving other modes of inheritance that can give rise to the clinical picture of epilepsy. The

relatively frequent occurrence of the condition in both parents and children, as well as the fact that consanguinity is not found with unusual frequency among the parents of epileptics.

A significant proportion of the children of people with epilepsy die at an early age from 'convulsions'. On the other hand, there are people with epilepsy in whom only very few or even a single seizure is observed in their lifetime; consequently, there are likely to be many carriers of the predisposition who never experience a seizure in their lifetime. This, of course, makes it very difficult to determine the pattern of inheritance. Lundborg's myoclonic epilepsy (see p. 222) is almost certainly recessive; indeed, it also constitutes a form of 'genuine epilepsy'. Genuine epilepsy, too, is by no means an idiotypical entity. The fact that there are more than twice as many males as females among people with epilepsy – even in the first two decades of life, when alcohol and other harmful factors cannot account for this difference – suggests that there may also be sex-linked recessive predispositions to epilepsy.

So-called migraine bears certain similarities to epilepsy, manifesting itself primarily in sudden, severe, unilateral headaches. As the condition can be traced through several generations in some families, it usually appears to involve dominant genetic predispositions; and since women are affected more frequently, a sex-linked dominant pattern of inheritance should be considered.

Bilateral headaches, which persist more or less throughout a person's entire life without any other mental disorder, also appear to have a hereditary basis: 'familial headache'.

Among actual mental illnesses, the significance of hereditary predisposition is most pronounced in the so-called manic-depressive or manic-melancholic disorders. This refers to a group of mental disorders characterised by more or less severe disturbances in mood.

These include m e l a n c h o l y, which manifests itself in a complete inhibition of mental activity due to a profound sense of sadness, furthermore, mania, in which mental life is thrown into confusion by unbridled, elated excitement; and cyclical or periodic mental disorder, in which periods of manic, melancholic and normal states alternate. Even states of simple melancholia or mania tend, after a shorter or longer period, to give way once more to a normal or near-normal mental state. There is therefore no permanent disruption of mental life. As a rule, even during relatively healthy periods, milder constitutional anomalies are evident, which may persist on their own without ever leading to severe mental disorders. Thus, a distinction is made between a melancholic, a manic, a cyclothymic and an irritable constitution.

If one were to summarise the entire group of manic-melancholic mental disorders under a single name, one might well speak of parathyme (mood disorder). In many cases, parathymic mental disorders can be traced back through several generations. It may therefore be assumed that at least some of the predispositions within this group—which, incidentally, certainly do not simply correspond to any of the clinical pictures mentioned—are dominantly inherited. In the families of parathymic mental patients, one usually also finds parathymic psychopaths who need not necessarily develop more severe mental disorders.

It appears that the predispositions to parathymic psychopathy generally exhibit dominant inheritance and that, in individuals affected by them, episodes of severe parathymic insanity may occasionally be triggered by causes that are not yet fully understood. The fact that approximately twice as many women as men are observed to suffer from parathymic mental disorders suggests, to a certain extent, a sex-linked dominant pattern of inheritance for some of the genetic predispositions to parathymic conditions. This would also be consistent with the finding, according to Reiss, that similar disorders are found about 1½ times more frequently in the mother than in the father of the patient.

An extremely important, psychologically induced clinical picture arising on a hereditary basis is h y s t e r i a. The term 'hysterical' is primarily applied to certain seemingly physical medical conditions for which, however, no physical cause can be identified and which may suddenly disappear without a trace or change in nature. Thus, hysterical paralysis of the limbs may occur, as well as numbness in localised areas of the body, all manner of pains, blindness, deafness, muteness and a wide variety of other symptoms. Very characteristic are peculiar convulsive fits accompanied by loss of consciousness, which may outwardly resemble epileptic fits. I would like to define hysteria as a more or less unconscious and involuntary imitation of clinical pictures.

It is not necessary for the hysteric to have previously observed the imitated clinical picture; rather, they present a picture as they imagine a particular medical condition might appear. Hysteria can be compared to mimicry; a mimic animal, which impresses its pursuers by imitating another poisonous or otherwise dangerous animal, presumably does not realise that it is imitating. Similarly, the partridge hen, which feigns a wing injury and thus diverts a predator's attention away from her chicks, probably does so instinctively and unconsciously. The same applies to the 'playing dead' behaviour of many animals, particularly insects.

The triggering of individual hysterical symptoms is brought about by a keen desire for an object or goal, for the attainment of which the hysteric, more or less unconsciously, perceives the presented state of illness as a suitable means. Thus, during the war, the desire to escape enemy fire and return home led to all manner of hysterical symptoms, particularly often to persistent states of trembling and fidgeting. In the case of pension hysteria, it is the desire to obtain a pension that gives rise to the clinical pictures deemed suitable for this purpose. However, the link with the desired goal is always more or less unconscious to the hysteric; this distinguishes hysteria from malingering. In everyday life, it is often the desire to evade unpleasant duties, or to arouse pity, or to make oneself seem interesting, or to cast blame on someone else (who is supposed to be to blame for the illness). This is why the imitation of epileptic convulsions – which, as experience shows, always make a great impression on laypeople – is so popular amongst hysterics. Epilepsy was, after all, long regarded as a 'sacred disease' (morbus sacer). When a hysteric lies there convulsing, any anger directed at them for having neglected their duties usually turns into pity or superstitious fear. The striking image of chorea is also readily imitated. But it is not only physical but also mental states of illness that lend themselves to hysterical mimicry; melancholic states are used to evoke pity, whilst states of agitation are employed to attempt intimidation. Because the illness is a means to achieve a fervently desired end, it is incorporated into the will.

Whilst the triggering of hysterical symptoms occurs through external events that give rise to intense desires, the constitutional basis is to be found in a hereditary, abnormally strong susceptibility of ideas, judgements, feelings and even sensations to the influence of more or less unconscious desires. This abnormal susceptibility to desire need not manifest itself solely in the imitation of clinical symptoms; far more often it simply leads to the repression of unpleasant ideas or memories from consciousness, or, conversely, to all manner of delusions or wish-fulfilment illusions. Thus it happens time and again that individuals with a corresponding predisposition imagine, without any reason, that a particular person of the opposite sex—or even several at once—is in love with them.

Since I consider it appropriate to restrict the term 'hysteria' to the involuntary mimicry of clinical symptoms, as this also best corresponds to medical usage, I propose the term 'orgoristia' for the underlying pathological will-determination, which may also manifest itself in entirely different ways.[D]

Of course, not every form of wish-determinism is to be regarded as pathological. Even healthy people are inclined to believe what they wish; yet the insights gained from experience and logical thinking do set certain limits to this wish-determinism. A certain

The soul's capacity for volitional determination is evidently consistent with the preservation of both the individual and the collective. It is the foundation of faith, love and hope. It enables views and volitional orientations—which have been proven to be consistent with the preservation of a community—to be incorporated into the will of its individual members. It is therefore also a physiological basis for custom, morality and religion. It may also be the case that, under certain circumstances, even the involuntary imitation of pathological states can occur without a hereditary predisposition to pathological volitional malformation. In the case of those people I have in mind, however, volitional malformation has degenerated to such an extent that it impairs both their own survival and that of society. And it is only this pathologically heightened volitional determinism that I wish to designate by the term 'orgorism'. The anti-preservation nature of orgorism becomes clearly apparent, for example, when – as is quite common – orgoristic individuals repeatedly put their own family in the wrong in front of strangers, or when, in political life, they seek in every way to denigrate their own state and their own people, and, on the other hand, in the fact that they plunge themselves or their people into disastrous undertakings contrary to all reasonable calculation, because they are unable to distinguish, or can distinguish only to a limited extent, between their illusory desires and the actual facts or possibilities. They therefore constitute the majority of leaders in political, religious and anti-religious excesses. Given this immense and usually disastrous influence of orgorism on the life of the race, a more detailed discussion of it is warranted.

There can be no doubt that orgorism is essentially rooted in the genetic makeup. It is likely, however, that there are a whole range of different pathological genetic predispositions which give rise to abnormal susceptibility to desire. According to my personal observations, such predispositions generally appear to be dominant. Depending on how a predisposition to orgorism interacts with other character traits, however, a quite varied picture may emerge; thus, some distinctly orgoristic individuals are amiable and harmless, whilst others are malicious and dangerous. With regard to hysteria, it is reported that the majority of sufferers are descended from psychopathic parents. Genuine hysterical disorders, i.e. the mimicry of other pathological states, do not, however, appear to occur regularly among the parents. The orgoristic predisposition as such is, of course, generally not recognised by laypeople; in reality, therefore, its prevalence among parents is likely to be considerably higher than the statistical data suggest.

It is usually stated that hysteria is several times more common in women than in men—up to ten times as common. However, one must not infer from this that the predisposition is distributed in exactly the same way; for men, feigning illness is, in times of peace, simply not usually a suitable means of asserting one's will. The experiences of the Great War have shown that the orgoristic predisposition is also much more common among men than was previously assumed; it manifested itself in hysterical symptoms in many thousands of them. K r a e p e l i n, drawing on data from psychiatric clinics—which usually admit only particularly severe cases of hysteria—found a ratio of only 1 : 1.9 to 1 : 2.3 to the disadvantage

the female sex. If one assumes that, particularly in severe hysteria, external triggers play a lesser role compared to hereditary predisposition, and that these figures therefore come closer to the ratio of predisposition in both sexes than other figures, one would have to consider the existence of sex-linked dominant predispositions to orgorism.

Paranoid mental disorders have close links to orgoristie. One speaks of Paran oia or madness when a system of unshakeable delusions gradually develops without any particular impairment of the otherwise clear thinking. There are all manner of transitions to mental health. Paranoid psychopaths are characterised by a peculiar mistrust of their surroundings, which tends to go hand in hand with a more or less conspicuous overestimation of their own abilities. They feel misunderstood, treated with hostility, disadvantaged and disregarded. Quarrelsome individuals, who, on the basis of perceived or—more often—actual injustice, make extremely persistent and protracted efforts to assert their legal claims, are also usually classified within this group. In any case, the various forms of paranoid psychopathy are also decisively hereditary in nature.

Whether they can be clearly distinguished from orgoristic psychopathy is questionable; whilst their manifestations are more persistent and less erratic, wishful thinking undeniably plays a significant role as well. In the case of orgoristic researchers, for example, the burning desire to be regarded as a man of importance tends, sooner or later, to lead to the conviction that they have made an epoch-making discovery. Under favourable conditions and given a corresponding predisposition, the realisation may well take hold once more that it was, after all, nothing so earth-shattering, or that it was even a mistake. This cycle may repeat itself several times. In the case of strong-willed individuals, however, it is not uncommon for these convictions to become permanent, particularly under unfavourable external conditions, for example, whilst in a position of authority. Particularly when defending themselves against criticism, orgoristic researchers may become increasingly fixated on a single theory, until eventually their entire scientific identity stands or falls with their supposed—or, more often than not, actual—discovery. Their colleagues usually regard them as paranoid or, in cases of frequent oscillation between discovery and disappointment, as 'manic-depressive'.

Orgorism is also related to parathymic (manic-melancholic) mental disorders. If only those parathymic disorders in which triggering by external influences plays no role are to be included, then not many of the milder disorders that are usually classified in this group are likely to remain. Hysterical symptoms are often observed directly in parathymic disorders. On the other hand, it is known that abnormal mood swings frequently occur in orgoristia ('hysterical predisposition'). It is therefore likely that there are certain idiotypical relationships between the groups of parathymic, orgoristic and paranoid mental disorders, which are, after all, distinguished solely on the basis of their clinical manifestations. One must bear in mind that there are pathological hereditary predispositions which, depending on

interaction with other factors and, perhaps, also depending on external conditions, may manifest as symptoms characteristic of one of the three groups.

Neurasthenia or nervousness consists of an abnormally high degree of mental fatigue and the associated abnormally high irritability. As this is a mental anomaly, the term psychasthenia (according to Janet) would actually be more accurate. In contrast to mental deficiency, referred to as 'feeble-mindedness', psychasthenia is a mental weakness of an entirely different kind. Mental alertness is, on average, rather heightened, but stamina is low. A distinction is usually made between constitutionally determined nervousness and neurasthenia acquired through overexertion; in both cases, however, idiotypical and parakinetics causes certainly interact. Whilst nervous exhaustion may be triggered by the ordinary stresses of working life, hereditary predisposition is of greater practical importance; conversely, when it is caused by exceptionally gruelling exertions and worries—these external causes. Hereditary predispositions to psychasthenia can certainly be of quite different kinds. Thus, it seems that an unusually strong mental activity, or an excessively uninhibited flow of thoughts, can lead to mental exhaustion, most readily in those of a frail physique. Hence, psychasthenics often exhibit an asthenic constitution; yet there are also psychasthenics of robust build.

It has been established that around one third of neurasthenics are descended from psychopathic parents. If the mental constitution of the parents were always known in detail, however, the figures would certainly be much higher. I have the impression that the predisposition to psychasthenia is dominant in many cases. Furthermore, there will of course also be predispositions with other modes of inheritance.

There are close links between psychasthenia and orgoristia. Both conditions are characterised by a high degree of emotional instability. The symptoms of both conditions often occur together. Among neurasthenics, there are roughly twice as many men as women. The greater demands that life tends to place on a man's mental capacity apparently cause abnormal fatigue to manifest itself more frequently in him. On the other hand, a hereditary predisposition that leads to nervous exhaustion in men presumably manifests itself in women as hysterical symptoms. Furthermore, psychasthenia is related to various other psychopathies, though none of these are nearly as common and are therefore not as significant as the forms mentioned. We can all the more refrain from a more detailed discussion of these psychopathies here, given that their idiotypical special status or, on the other hand, their affiliation with the

types discussed has by no means been clarified to date. The only certainty is that the decisive cause lies primarily in the genetic makeup.

It is not uncommon to speak of a specific 'degenerative insanity' and its heritability. What is designated as such generally encompasses severe forms of orgoristia ('hysterical character'), paranoid psychopathy and related anomalies. However, it is not clear why, for example, schizophrenia or epilepsy should not be classified as degenerative conditions. We understand the term 'degeneration' in the broadest sense to mean the emergence and spread of pathological hereditary traits. We cannot therefore recognise 'degenerative insanity' as a clinical or biological entity.

Nor is the term 'm o r a l i n s a n i t y' ('moral imbecility', 'moral insanity') suitable for defining a specific type of mental disorder. The concept of
'moral' is not a scientific one; and indeed, a wide variety of pathological conditions are lumped together under the heading of 'moral insanity'. If one is determined not to abandon the term altogether, then one should at least use it to refer to clearly defined anomalies. Thus there are people who, without any other mental disorder, are characterised by boundless emotional cruelty; they are 'born criminals', and their lives are therefore more or less filled with crime and prison sentences. Naturally, this serious mental abnormality—which, incidentally, is not common—is overwhelmingly of idiotypical origin. More common are individuals who, without actually being malicious, lack so-called 'inhibitions'; who, yielding to immediate sensory impressions and without any conception of the consequences, simply follow their respective instinctual impulses—who, for example, simply steal whatever seems desirable to them at the time, who have no sense of truth, and who also act on their sexual urges without regard for the consequences. This anomaly, too, is essentially hereditary; such individuals are difficult, if not impossible, to educate.

Anomalies of the sexual instincts are also to be included amongst psychopathies. A pathological intensity of the sexual instinct clearly runs in families; conversely, an abnormal weakness or complete absence may also occur. A large proportion of women lack a true sexual drive, whilst their maternal instincts may well remain intact. Since this need not impair reproduction in women, the absence of the sexual drive can probably only be regarded as genuinely pathological in men. It has been suggested (e.g. by Schallmayer) that human beings do not possess a true reproductive instinct at all, because reproduction is merely a consequence that is not usually sought when the sexual instinct is acted upon. It does not, however, seem to me to be part of the nature of an instinct that its goal should be consciously sought. Animals are certainly unaware of the aims of many of their drives, and yet the mating drive is unconsciously directed towards reproduction. It should also be borne in mind that, in particular, a normal woman without children does not tend to find complete fulfilment. In my view, therefore, one can also speak of a reproductive drive in humans, which encompasses a number of individual drives.

The most significant anomaly of the reproductive instinct is so-called homosexuality, in which the sexual instincts are directed towards persons of the same sex. In some cases, this is probably to be understood as an expression of sexual intermediate stages (cf. p. 182), since homosexual individuals more frequently display physical characteristics reminiscent of the opposite sex than do others. In a larger proportion of cases, however, it is precisely the contrast between physical constitution and the psychological orientation of the drives that is striking. Homosexual tendencies are not uncommon amongst several members of the same family. Yet even without any particular hereditary predisposition, homosexual feelings appear capable of developing, particularly on the basis of orgorism, in which the drives

and feelings are easily diverted and influenced by experiences. Hereditary predisposition therefore plays a greater or lesser role in individual cases, depending on external influences. In any event, however, homosexuality is by no means a normal variant, as some of its defenders tend to portray it, but rather something distinctly pathological, because it impairs the preservation of the species to a significant degree.

3. Determining the hereditary transmission of pathological predispositions.

It is by no means always easy to determine whether a condition is hereditary or not. If an anomaly occurs in several siblings or across several generations of a family, one must certainly consider the possibility of a hereditary cause; however, this is not proven by the mere fact of familial clustering. The common cause of a disease affecting several family members may, in fact, also lie in shared external harmful factors, for example, infection with the same pathogen or the effects of toxins affecting several family members simultaneously. However, one must not overestimate this risk of misinterpretation. In a great many cases, the idiotypic nature of an anomaly can be deduced from the nature of the anomaly itself, for example in the case of malformations that occur in the same way on both sides of the body or symmetrically on the hands and feet. If a child has the same two fingers fused together on both hands, and the same is true of one of the parents, then no one would rightly doubt the hereditary nature of this anomaly.

> Far more frequently than hereditary predispositions, external causes of a condition tend to be hastily assumed, given the almost universally prevalent overestimation of environmental influences. Precisely when only some members of a family are markedly affected by a condition, whilst the rest are not, this suggests—contrary to the view that still generally prevails—with a high degree of probability that the condition is at least largely due to hereditary factors. Where, despite careful investigation, external causes cannot be identified or even made plausible, one must always consider a hereditary basis. There are diseases which, without any significant contribution from external causes, appear to
> arise 'spontaneously', are termed 'idiopathic' or 'genuine' conditions; that is to say, the same root terms have been adopted as those used by modern research into heredity to denote hereditary (idiotypic, genotypic) predispositions. Idiopathic or genuine conditions are therefore hereditary conditions. Determining heredity is made very difficult when, in the development of a disease, external influences play a significant role alongside the hereditary predisposition. In such cases, it is essential to demonstrate that the external harmful factors do not, on their own, cause the disease, or at least do not cause it with the same severity as when a corresponding hereditary susceptibility is present.

Even when there is no doubt as to the idiotypical nature of a condition, it is often still very difficult to determine whether its development

is essentially determined by just one or by several genetic units. Nor is it always easy to clarify the mode of inheritance of a single predisposition. For research into the genetic predispositions of animals and plants , systematic breeding experiments are available, which, with sufficient care and effort, ultimately always lead to a clear conclusion. In humans, however, such breeding experiments are naturally not possible. To determine the inheritance patterns of human traits, therefore, in addition to inferences by analogy from animal experiments, statistical research is the primary method, which can be conducted either on an individual statistical basis (casuistic) or on a population statistical basis (summary). Individual statistical research into heritability investigates specific traits within individual kinship groups. Population-based heredity research, on the other hand, compares the average characteristics of a group of people selected on the basis of specific traits with those of their offspring or other relatives. It is often advisable to analyse the data obtained through individual-based research using population-based methods as well.

In individual research into the inheritance of pathological predispositions, it is very important that, as far as possible, the entire immediate family of a patient—who is the subject of the investigation—is examined. Staff must be made aware that it is not only the patients but also the healthy family members and the children of the healthy individuals who are to be identified. Furthermore, the age of all family members at the time of the examination should always be recorded, particularly in view of the fact that certain hereditary conditions do not manifest until middle or later life.

A particular risk in such studies is that the data collected often represents a biased sample and is therefore not typical of the average population. There is a strong tendency to publish ‘interesting cases’ of heredity; and cases are regarded as such particularly where a large number of family members are afflicted with anomalies, which, however, need not have anything to do with heredity as such, but may be due to chance (cf. p. 161). Unremarkable cases of a condition are therefore often just as significant as those that appear particularly

interesting ones. Above all, individual cases, such as the one shown in Fig. 27, must not be omitted on the grounds that 'heredity is not present'. It is by no means necessary that a hereditary condition found in one person must also be found in other members of their family. Particularly in the case of recessive conditions, this will usually not be the case, given the small size of most families in our population.

The small size of the human family gives rise to a major difficulty for research into heredity: in most cases, it is simply not possible to establish, with even a degree of reliability, a generally valid numerical ratio amongst the members of a family. One is therefore compelled to aggregate the data from various families, but in doing so runs the risk of counting together idiotypically different anomalies that are only superficially similar, thereby rendering the overall result questionable.

> Some have even sought to see in the small number of children a counter-argument against the validity of Mendel's laws in humans; but this is out of the question. In animals and plants, too, Mendel's ratios arise solely from the combination of probabilities that exist for the genetic constitution of each offspring given the specific characteristics of the parents. But this applies in exactly the same way to humans as well. The greater difficulty in determining the figures due to the small number of siblings therefore does not represent a fundamental difference. The popular objection that one should not draw conclusions about humans from experiments on animals and plants is entirely inappropriate in the context of Mendel's law. If this law is found to hold true for butterflies or flies just as it does for rabbits or chickens, or even for plants — and this is a fact — then an inference regarding humans is entirely permissible; for humans are biologically incomparably closer not only to rabbits and chickens, but also to butterflies and flies, than the latter are to plants. Of course, however, one must not simply transfer the pattern of inheritance of a particular trait observed in animal experiments to humans without further consideration; for, with regard to individual genetic traits, different animal species naturally behave differently. Thus, the white colour of albino rabbits is inherited recessively, whereas that of grey horses appears to be dominant.

Until just a few years ago, the often difficult task of verifying Mendelian numerical ratios was necessary to establish the validity of Mendel's laws for humans at all. Fortunately, this is no longer necessary today; the determination of these numerical ratios therefore now serves more to distinguish between the various possibilities of inheritance and to assess whether, in the case of a condition, external influences are at work in addition to the genetic factor;

for if that is the case, the proportion of affected siblings will fall more or less short of the expected Mendelian figure. In order to establish whether a condition is hereditary at all, it is first necessary to show that, amongst the siblings of those affected, there is a higher proportion of affected individuals than is otherwise found in the general population. Identifying dominant inheritance generally presents no difficulties; it has been discussed above using several examples (see, for example, p. 160). It is more difficult to distinguish between simple dominant and sex-linked dominant inheritance; in the case of a few family trees, this distinction is often impossible to make at all. A greater prevalence of affected females suggests that a dominant trait is sex-linked; transmission from father to son rules this out (see p. 165). Identifying sex-linked recessive inheritance generally presents no difficulties, as in such cases almost only men are found to be affected, whilst transmission from father to son does not occur. (See p. 151).

> When we describe a pathological genetic trait as dominant, this, strictly speaking, actually implies the assumption that the trait would manifest itself in the homozygous state in exactly the same way, or at least very similarly, as in the heterozygous state. In fact, however, we know almost all those pathological genetic traits which we regard as dominant only in the heterozygous state; and it is not unlikely that most of them would present quite differently in the homozygous state—and presumably in a much more severe pathological form. Of nine apparently dominant pathological idiovariations that occurred in Morgan's fly lines, only three were identical in both the homozygous and heterozygous states, whilst six were no longer compatible with life at all in the homozygous state. Strictly speaking, therefore, such cases do not actually involve dominance, but rather intermediate behaviour or incomplete dominance (cf. p. 27). If this was not taken into account when discussing the individual pathological traits, it was done in the interests of simplicity of presentation and because, in humans, such traits practically never occur in a homozygous state. Nevertheless, one must bear this possibility in mind.

The most difficult aspect is the reliable identification of recessive inheritance, particularly when more than one pair of alleles is involved or when external factors play a part in triggering a condition. In the case of simple recessive inheritance, by far the most common scenario is that both parents of an affected individual are healthy and merely carry the allele in a masked state. Since, on average, only a quarter of the children of such parents develop the condition, in many families where both parents carry this genetic trait, there will be no affected child purely by chance; and since in these families the presence of the allele cannot be detected either in the parents

nor in the children, such families will escape statistical recording. However, if only those families are included in the count where, by chance, at least one child is affected, this inevitably results in an inflated percentage of affected individuals. Nevertheless, it is possible, under certain circumstances, to determine the correct ratio with a high degree of accuracy. This is achieved using the sibling methods described by Weinberg, the principle of which may be illustrated by an example.

If the probability that a particular child will develop the condition, given the parents' genetic constitution, is ¼, then, assuming there are two children in each family, on average only in every 16th marriage will both children be affected. The distribution of healthy and affected children in marriages with two children is given by the binomial formula $(1\ k + 3\ g)^2 = 1\ kk + 3\ kg + 3\ gk + 9\ gg$. It is therefore represented by the following diagram, in which each pair of circles arranged one below the other always represents two children from a single marriage.

Fig. 64.
Probable distribution of healthy and affected children in marriages with two children, where both parents carry a recessive pathogenic gene.

The ratio of affected to healthy siblings is 1 : 3, as is consistent with recessive inheritance. However, if the survey only records those families in which there is at least one affected child, the last nine families are omitted from the survey and the ratio obtained is 8 : 6, which does not at all correspond to recessive inheritance, on the basis of which the composition of the families was constructed. The correct ratio, on the other hand, is obtained by comparing the number of affected siblings of affected children with the number of healthy siblings of affected children. In the first family, each of the two children has one affected sibling (the second is an affected sibling of the first, and the first is an affected sibling of the second). In the remaining six families, where affected children are present, these children have only healthy siblings, namely six in total. If one compares this number with that of the affected siblings of affected children—namely two—one obtains the correct ratio of 2 : 6 = 1 : 3, as corresponds to recessive inheritance with a probability of ¼ for each child. A completely analogous result could, of course, be demonstrated for families with a larger number of children.

Just as with recessive inheritance, the sibling method is also applicable to dominant inheritance; indeed, it can be used wherever the aim is to determine a numerical ratio amongst siblings, for example, to decide whether boys occur more frequently in some families and girls in others than is consistent with the general probability distribution.

However, an essential prerequisite for the applicability of this sibling method is that either the population under study truly includes all families with carriers of the trait to which the research relates, or that the actual data has the same composition as the

total population, meaning that a representative 'selection of families' according to the characteristic is available. However, this is usually not the case. As a rule, it is not feasible to survey an entire population; instead, the research takes as its starting point individual persons who exhibit the characteristic, for example, those who seek treatment from a particular doctor or at a clinic. Since it can be assumed that, in this context, every individual exhibiting the characteristic generally has the same probability of being included in the study, a family in which individuals exhibiting the characteristic are present has a greater probability of being included the more such individuals it contains. Such a selection of families, which is based on individuals possessing the trait, is called 'individual selection', and naturally includes a relatively greater number of families with many carriers of the trait than 'family selection'. The sibling method, as described, would therefore still yield figures that are too high if applied to individual selection.

In the diagram above, family 1 is twice as likely to be included in an individual selection as any of the following six families. In an individual selection drawn from a population composed in this way, therefore, for every family with two affected children, there would be only three with one affected child (whereas in family selection, the ratio would be one in six); consequently, even the sibling method described above yields values that are still too high in the case of individual selection – in the hypothetical example, 2:3 (instead of 1:3).

For individual selection—as approximated by data derived from patients of a doctor or a hospital—the Weinberg 'proband method' is far more suitable; this involves counting only the siblings of those individuals carrying the trait from whom the family survey originated ('proband's'), whilst excluding the probands themselves.

Among the siblings of the probands in families 1–4, which correspond to 'individual selection'—one affected individual is matched by three healthy ones; this thus yields the 1:3 ratio typical of recessive traits, with a probability of occurrence of ¼ for each child. The term 'proband' originates from genealogy and originally referred to individuals whose ancestry was to be investigated, i.e. the starting points of a genealogical inquiry. Individuals who carry the trait but are recorded solely because of their kinship to probands are termed 'secondary cases'. Siblings of probands may themselves also be probands, namely when they are themselves directly recorded, for example, if they themselves were admitted for treatment as patients. As the recording of all carriers of a trait within a population as probands progresses, individual selection gives way to the comprehensive recording of all families, and the proband method gives way to the more specific sibling method.

When determining whether a condition is caused by a recessive genetic trait or not, establishing the frequency of consanguinity among the parents can be of considerable assistance.

> Hammerslag, for example, found that of 107 children born deaf, 42 – that is, about 40 per cent – were born to consanguineous parents, whilst of 130 children who became deaf later in life, only 13 – or about 10 per cent – were born to consanguineous parents. It is, however, quite possible that recessive genetic factors may also play a role in some cases of postnatal deafness; for the general frequency of consanguineous marriages is, after all, considerably lower than 10 per cent.

The rarer the recessive condition in question is within the population, the higher the frequency of consanguineous marriages among the parents is likely to be. If a particular recessive predisposition to disease were present in a latent form in just a single family, two such predispositions could only be brought together—and thus become apparent—through consanguineous marriage; in this hypothetical extreme case, 100 per cent of those affected would therefore be the product of consanguineous marriages. If, on the other hand, a recessive predisposition to disease is relatively widespread in a population, two such predispositions will naturally often come together even without consanguineous marriage, and the frequency of consanguineous marriage amongst the parents of those affected will be correspondingly lower. In the case of very widespread recessive traits, such as the trait for blue eye colour, one would therefore not expect to find a demonstrably increased frequency of consanguineous marriages amongst the parents. However, pathological traits with recessive inheritance will, of course, never be anywhere near as common; and if, even with a large sample, one cannot demonstrate an above-average frequency of consanguinity among the parents of certain patients, this strongly argues against the condition being caused by recessive genetic factors.

> It can be very difficult to determine whether a condition is simply recessive or whether it is caused by the interaction of several different genetic factors. For example, one might imagine that two different genetic factors must be present in a homozygous state for a particular condition to manifest itself; the condition is then referred to as doubly recessive. If one of these two alleles were widespread in a population, the other would exhibit simple recessive inheritance. If, on the other hand, both are relatively rare, there will be fewer affected individuals amongst the siblings of affected individuals than would be the case with simple recessive inheritance. If, for example, an affected individual has the genotype a a b b, they may be descended from two outwardly healthy parents with the genotype A a B b. They may then, amongst other possibilities, have healthy siblings with the genotypes A a b b or a a B b. In such cases, factor A would act as a suppressor in relation to the pathological predisposition b b, and factor B as a suppressor in relation to a a. Some geneticists

are very inclined to assume the existence of such inhibitory factors, although it has not yet been possible to actually demonstrate them in specific diseases. In any case, it must be insisted that such factors are not simply 'assumed', but that the conclusions arising from such assumptions are actually calculated numerically and compared impartially with actual experience.

In the case of dominant inheritance, inhibitory factors are often posited to explain the frequently reported skipping of one or more generations in the inheritance pattern. It is thus assumed, for example, that a predisposition C can be prevented from manifesting by an inhibitory factor D, so that an individual with the genotype C c d d would be affected, whilst one with the genotype C c D d would be healthy. In a population in which factor D did not occur, predisposition C would therefore simply exhibit dominant inheritance; if, on the other hand, factor D were widespread in the population, the inheritance of the condition would appear to be interrupted more or less frequently, making it difficult to distinguish it from recessive inheritance. In fact, there would then be a certain similarity to recessive inheritance. For if the C factor were widespread throughout the population, the very absence of the D factor would be decisive for the occurrence of the condition, and this would simply be recessive, in that affected individuals would be C C d d. If both factors were present in moderate frequency within a population, the condition would therefore exhibit a pattern of inheritance combining dominance and recessiveness, in that its occurrence would depend on the combination of a dominant factor with a recessive one in a homozygous state. However, nothing of the sort has actually been demonstrated to date.

Just as the hypothesis of inhibitory factors is popular, so too is that of triggering factors. For example, a dominant gene E is said not to be sufficient on its own to cause a condition, but only when the factor F is present at the same time. In a population where F were widespread, E would therefore exhibit simple dominant inheritance; conversely, in a population where E were widespread, factor F would determine simple dominant inheritance of the condition in question. Both factors would be equally indispensable for the onset of the condition, and it would depend solely on the relative frequency of the two factors as to which would be regarded as the 'actual' pathological genetic predisposition and which as the triggering factor. An affected individual would generally have the genotype E e F f, and if the other spouse were normal, namely e e f f, then one quarter of the children would again be expected to have the genotype E e F f and thus the condition. Since in most cases the parents of an affected individual would be normal— — distinguishing this from recessive inheritance would therefore present difficulties . Nevertheless, in the case of conditions caused by two independent factors, the occurrence in one of the parents would be more frequent than with recessive conditions. Careful calculation of the theoretical proportions and comparison of these with the actual figures is therefore required. Blood relationship would not be as significant here as it is in the case of recessive inheritance.

Whilst we are of the opinion that inhibitory and triggering factors have often been assumed without sufficient justification, and that the interaction of many factors does not, in general, play as significant a role in human conditions as some researchers believe, we are nevertheless of the view that pathological genetic predispositions can manifest themselves in quite different ways depending on the other genetic predispositions with which they interact; and this view has, in fact, been expressed on several occasions during the discussion of individual conditions.

Research into heredity encounters particular difficulties when no clear distinction can be made between siblings into distinctly separate groups

, but where all manner of continuous transitions and merely quantitative differences in degree are found, as is usually the case, for example, with height. In such instances, research into heredity must, for better or worse, resort to the summary method and be content with determining the average similarity between different degrees of kinship with regard to the trait in question.

The correlation coefficient is usually used as a measure of average similarity; this is employed in particular by the so-called biometric school in the study of heredity. If two groups of individuals are completely identical with regard to a particular trait, the correlation coefficient is 1. This is the case, for example, in a pure-bred strain of albino rabbits between parents and offspring with regard to coat colour. If, on the other hand, two groups of individuals are no more similar to one another than any other individuals in the population, the correlation coefficient is 0. Space does not permit us to go into the very cumbersome calculations involved in determining the correlation coefficient in greater detail here; and we can all the more readily dispense with it as we are unable to share the high regard in which the correlation coefficient is held in England and America, and here and there in our own country as well. It is often overlooked that the correlation coefficient is meaningful only within a specific population. We can readily appreciate this simply from the concept of average similarity. Any ten individuals in our population are generally quite dissimilar to any other ten. If, however, these same two groups of ten individuals each were to live in a population consisting entirely of Black people, they would appear very similar to one another. Accordingly, in one case no correlation would be found at all, whilst in the other a high correlation would be found. Correlation analysis is not particularly well suited to assessing the heritability of traits that clearly distinguish their carriers from the rest of the population. If, for example, one were to calculate the correlation between parents and children with regard to a rare recessive trait such as albinism, one would find a coefficient of almost 0. If, on the other hand, the same trait were very common in the population, a much higher correlation coefficient would be found. However, a trait is, of course, no less 'hereditary' when it is rare than when it is common; and it is therefore obvious that the correlation coefficient—which yields very different values not only depending on the greater or lesser influence of external factors in triggering a trait, but also depending on its greater or lesser frequency—cannot be an exact measure of its heritability. In stark contrast to the appearance of precision conveyed by the complicated mathematical formula for the correlation coefficient, it is only useful as a rough guide to certain manifestations of heritability. Such an overview, however, can be obtained much more simply and clearly through graphical representation, as will be demonstrated here by way of an example.

Fig. 65.
Graphical representation of the relationship between the height of adult individuals and that of their parents, based on Galton's data.

If, for example, the heights of parents and children have been recorded in a large number of families with adult children, the pairs of parents can be divided into groups according to their height, and the average height of the children in each group can be determined. If one then plots the heights of the parent groups on the x-axis and the corresponding heights of the children on the y-axis in a coordinate system, the endpoints of the y-axis form a more or less irregular line,

which shows at first glance that as the parents' height increases, so does that of their children.

Unfortunately, it seems necessary to critically examine certain outdated prejudices that persist in the field of the heritability of diseases. A condition is often regarded as hereditary only if it was also present in the parents or other ancestors. When a condition is frequently observed in several siblings but not, as a rule, in the parents, one repeatedly reads that it occurs 'familially, but not hereditarily', even in cases where external causes clearly play no role in its development, such as in albinism. It should therefore be expressly pointed out once again that even recessive conditions, which generally give the impression of being 'familial, not hereditary', can be found in the direct descendants of affected individuals when people with the same predisposition marry, as can be demonstrated, for example, by a whole series of family trees of deaf-mute individuals. There is therefore no fundamental difference between 'hereditary' and 'merely familial' conditions.

Likewise, it is misguided to contrast 'direct inheritance' (from parents to children) with 'indirect' or 'latent inheritance' (from grandparents and other relatives). So-called 'direct inheritance' largely coincides with dominant inheritance, but not entirely, since the pattern of 'direct inheritance' can also arise in cases of recessive and polymeric inheritance. Furthermore, in the case of dominant inheritance of a predisposition, 'indirect heredity' may apparently occur if the predisposition requires external influences to be triggered, such as the predisposition to gout. The term

The term 'atavistic inheritance' has been used to describe the reappearance of traits from more or less distant ancestors, which is simply explained by the recombination of genetic factors. The term 'collateral inheritance' was often used when a condition was found not in ancestors or descendants, but in collateral lines, for example, among cousins. Furthermore, the term 'same-sex inheritance' was used when, within a family, only one sex (namely the male) was affected by a condition. We have come to recognise this phenomenon as one manifestation of sex-linked inheritance—though by no means the only one. Even 'crossed inheritance', which was contrasted with 'unisex inheritance', is in fact merely another expression of the same underlying law. Whilst the phenomenon whereby a hereditary condition manifests itself at roughly the same age in several members of a family has been termed 'corresponding inheritance', it should be borne in mind that every hereditary condition manifests itself for the first time at some point, whether before or after birth; there is therefore no reason whatsoever to regard this as a distinct form of inheritance.

Where conditions observed in ancestors and descendants were not identical but merely similar or even entirely different, the term 'disparate' or

'polymorphic' as opposed to 'uniform inheritance'. We now know that such cases cannot be explained by the 'transformation' of a genetic trait, but that individual genetic traits strictly retain their distinct characteristics, whilst they may present a different picture depending on their interaction with others and on external conditions. Incidentally, the accumulation of various hereditary anomalies and disorders is explained partly by repeated damage to the genetic material and partly by social selection, a subject which will be discussed in more detail later.

In any case, it seems to us that the time has come to finally consign all the various forms of inheritance that have been distinguished to the scrap heap. There is only one form of inheritance, and this is based on distinct units of the idioplasm, each of which has a probability of ½ of contributing to the constitution of a particular child.

4. The emergence of pathological hereditary traits.

All pathological hereditary predispositions must, of course, have arisen for the first time at some point. It is true that certain predispositions to disease can be traced back through numerous generations—for example, night blindness in a family tree spanning more than three centuries; recessive predispositions may also have been present in a population in a latent state for many generations before manifesting for the first time; but at some point, naturally, they too must have arisen anew. It goes without saying that the genetic material of the first humans did not yet contain all those hereditary predispositions to disease that are now widespread amongst present-day populations. Since there is no fundamental biological difference between pathological and normal traits, pathological hereditary traits naturally do not differ from so-called normal ones in terms of their origin. Since the genetic material must be conceived as determined by chemical and physical factors, it must also be capable of being altered by chemical and physical influences; and since individual genetic units, owing to their molecular structure, cannot exhibit gradual transitions, their alteration must also occur in more or less significant leaps or 'in bursts', through the loss, addition or rearrangement of molecules or groups of molecules. We now designate those chemical or physical influences which result in changes to the genetic material as i d i o k i n e t i c influences, and the process of causing such genetic changes or idiovariations itself as I d i o k i n e s i s.[E]

> The word 'idiokinese' is not synonymous with the term 'blastophthoria' (germ decay), as this term also referred to non-hereditary changes and, moreover, was restricted to harmful changes. We refer to the process of causing non-hereditary changes, whether in mature organisms or in germ cells, along with Siemens, as parakinesis; and we therefore also speak of parakinetical influences as opposed to idiokinetic ones.

In detail, our understanding of the conditions governing idiokinesis is still very incomplete; nevertheless, it has already been possible to artificially induce genetic changes in animal experiments. The American zoologist T o w e r caused
for example, beetles of the genus *Leptinortarsa* (Colorado or potato beetles)

to abnormally high temperatures (approximately 35°) during the maturation of their gametes. The offspring then largely exhibited deviating characteristics in colour, markings and size, which were subsequently found to be heritable. It is noteworthy that, in one and the same experiment under the same temperature conditions, several new forms arose alongside one another, alongside offspring that remained unchanged. When the same parent beetles were kept under normal living conditions during subsequent periods of gamete maturation, their offspring also returned to normal.

It is not unlikely that climatic influences may also bring about genetic changes in humans. This is supported, for example, by the fact that families of the Nordic race tend to die out in the tropics within a few generations due to the degeneration of their offspring.

Among our population, alcohol certainly plays a very special role as a cause of pathological hereditary traits. Among the offspring of alcoholics, all manner of ailments—which we must regard as idiotypically determined—are found in far greater numbers than is usual in the general population. About one third of all epileptics, the mentally retarded and the mentally deficient are descended from drinkers. Admittedly, in many cases the connection may also be such that the parents' (usually the father's) alcoholism was itself a manifestation of the same hereditary predisposition that manifests itself in the children as epilepsy or mental deficiency. Whether alcoholism is the cause or the consequence of mental inferiority cannot usually be determined in individual cases. However, it seems to me that there can be no reasonable doubt that alcohol can indeed have idiokinetic effects and that, in reality, it does so on a large scale.

> Continued alcohol abuse ultimately leads, almost without exception, to the destruction of the germ cells in the testicles. B e r t h o l e t found, among 100 corpses of alcoholics, 64 cases of complete atrophy of the germ tissue and only two cases of apparently normal testicles, whilst the remaining cases showed damage of a lesser degree. This explains why drinkers tend to become infertile sooner or later. It would indeed be a miracle if a toxin capable of completely destroying germ tissue did not, when its effect is weaker, occasionally result in alterations to the genetic material it contains.

Various attempts have also been made to demonstrate the harmful influence of alcohol on genetic material in animal experiments. Most of the experiments carried out to date, however, are of little value. The only evidence

to be the large-scale experiments conducted by the American professor Stockard. He exposed guinea pigs, which had previously produced normal offspring, to alcohol vapours daily for months (with the exception of Sundays). The animals did not fall ill as a result, but were also never entirely sober; their condition thus corresponded to that of many 'moderate' drinkers. Mating between such alcohol-exposed animals often proved fruitless; there were also many stillborn litters; the few offspring that were born usually died of convulsions, all of which occurred only rarely in non-alcohol-exposed control animals. Even when only the mother or only the father was exposed to alcohol, all manner of weaknesses and deformities occurred in the offspring; and when such degenerate animals were mated, pathological predispositions were also evident in the grandchildren and, likewise, in the great-grandchildren of the alcohol-exposed animals. By 1916, Stockard had already analysed the results of 164 matings of alcohol-exposed animals and, including the control experiments and subsequent breeding, 571 matings in total. Given the large numbers involved, these cannot be attributed to chance, and one must therefore conclude that corresponding damage to the genetic material caused by alcohol also occurs in humans.

In Germany, prior to the war, annual per capita consumption stood at around 10 litres of pure alcohol, a quantity equivalent to approximately 250 litres of beer. As this consumption is concentrated almost entirely amongst the male population of reproductive age, it is probably safe to assume that each man of reproductive age consumed 30 litres of pure alcohol annually, which corresponded to a daily consumption of about two litres of beer. In the cities, of course, alcohol abuse was even more widespread. In Munich, for example, per capita consumption stood at 287 litres of beer in 1907.

A distinction must be made between idiokinetic damage to offspring and purely parakinetetic damage, in which the genetic material remains unaffected. Thus, the frequent occurrence of miscarriages in families of alcoholics is probably attributable in part to direct poisoning of the foetus as a result of the mother's alcohol abuse; and the exceptionally high infant mortality rate is attributable in part to the disrupted family circumstances to which alcoholism tends to lead, but certainly also, in part, to damage to the genetic material. In cases of weakness in the offspring that are clearly caused by the father's alcohol abuse, purely parakinetics damage is highly unlikely; instead, genuine idiokinetic damage is generally to be assumed. Since the spermatozoon has a mass millions of times smaller than that of the egg, it cannot introduce any significant quantity of alcohol into the fertilised egg. Parakinetic after-effects, which might later be compensated for, can therefore, for the most part, only be attributed to the mother; and for this reason, it is precisely those experiments by Stockard—in which degeneration of the offspring occurred even when the father was the sole source of alcohol—that are of such great significance as evidence for the occurrence of idiokinetic damage caused by alcohol.

It is a matter of debate whether even temporary states of intoxication can give rise to degenerate offspring. It has been suggested that conception whilst intoxicated

is therefore disastrous because the temporary mental impairment of the intoxicated person is inherited. Naturally, this cannot be the case. It is not unlikely, however, that even mature sperm cells may be damaged by the alcohol circulating in the body, and that children with idiotypical impairments may result from such sperm cells once they have, so to speak, slept off their intoxication. B e z z o l a, based on the dates of birth of 8,186 mentally retarded individuals and idiots, has stated that a relatively large number of these were conceived during the carnival season and the grape harvest. E. H. M ü l l e r has found a similar deviation from the normal distribution of births among 847 epileptics. A connection is not unlikely, but cannot be regarded as proven on the basis of the evidence available to date. The fact that mature sperm cells in the body can be affected by alcohol has been established by experiments conducted by Cole and Davis. When a female rabbit was mated in quick succession by two males of different breeds, one of whom had been given alcohol, the resulting offspring were descended only from the male who had not been given alcohol. If, on the other hand, only the alcohol-treated male was allowed to mate, he was also able to produce offspring.

Apart from alcohol, a whole range of other poisons may be considered as idiokinetic causes, particularly those used for industrial purposes and which therefore often give rise to poisoning in industrial settings. These include lead, mercury, phosphorus, tobacco, carbon disulphide, aniline and related substances. Animal experiments have also been carried out with regard to lead. Professor Cole of the University of Wisconsin had female rabbits mated simultaneously by a male of a different breed that had been treated with lead and one that had not been pre-treated; it was found that the offspring of the male treated with lead were weaker and more prone to illness than their half-siblings from the same litter.

The idiokinetic effects of a number of medicinal substances, particularly protozoacides such as quinine, mercury, iodine and arsenic, cannot be ruled out. In animal experiments, even relatively small doses of free iodine – which otherwise have no noticeable effect on well-being – cause temporary infertility, whilst larger quantities result in permanent infertility. The poisons mentioned are, after all, all used to kill animal pathogens in the human body, e.g. the causative agents of malaria and syphilis; and the fact that human cells can also be killed as a result follows from the fact that these poisons can also kill the foetus in the womb. However, it is illogical to assume that germ cells are thereby always either completely destroyed or remain unharmed. Between these two possibilities lies, rather, the possibility of more or less extensive damage to the cells and their genetic material.

Of course, in practice, the medicines mentioned are nowhere near as significant for idiokinesis as, for example, alcohol. It is, of course, completely beyond my intention to oppose the use of poisons in medicine in general, as the so-called
'naturopaths' do. The substances mentioned are among our most effective and indispensable remedies. Nevertheless, the doctor should at least bear in mind the possibility of idiokinetic damage and should only prescribe the aforementioned remedies in large quantities or over a long period if it is truly necessary.

If we consider, in summary, the poisons from which we must primarily suspect harmful idiokinetic effects, it becomes apparent that they also exhibit a number of similarities in their other effects. All result in complete physical deterioration (cachexia, marasmus) following prolonged exposure; thus, alcohol-induced, lead-induced, mercury-induced, iodine-induced and arsenic-induced cachexia are known. This apparently involves damage to the idioplasm of the body's cells; for as long as such damage has not occurred, all cellular disturbances can be rectified. All the poisons mentioned also result in severe nervous disorders following prolonged exposure, usually in the form of multiple cases of neuritis accompanied by tremors and visual disturbances; the optic nerve in particular is easily affected by these substances; alcohol, nicotine, arsenic, lead, mercury, carbon disulphide and quinine frequently cause severe visual disturbances and even complete blindness. The central nervous system appears, in its sensitivity to toxins, to behave in much the same way as germ cells. It must therefore be assumed that all narcotics have an idiokinetic effect when exposed to them chronically. Another feature common to all the toxins mentioned is that they can lead to the death of the foetus in the womb and thus to miscarriage.

It can be assumed with certainty that X-rays, and likewise the radiation from radioactive substances—which are, after all, of a similar nature—have an idiokinetic effect. Oskar Hertwig irradiated sperm and egg cells of amphibians with radioactive substances and found that even in those experiments where only the spermatozoa were irradiated, the individuals resulting from the fertilisation of normal eggs with such spermatozoa exhibited all manner of malformations and states of weakness. He achieved very similar results by exposing mature spermatozoa to chemicals (methylene blue, chloral hydrate, quinine), and he comments: 'The experiments described, involving both radioactive and chemically active substances, have provided irrefutable experimental proof that they can permanently alter the idioplasm of the germ cells.' Furthermore, O. Hertwig concludes, in my view quite rightly, "that radium or X-rays can alter egg and sperm cells even whilst they are still within the gonads, just as they can after their isolation in the experiment".

Morgan and his students obtained some new idiovariations from cultures of the fruit fly (Drosophila) that had been irradiated with radium; however, this result could not be achieved consistently. This is, however, to be expected. The rays apparently only occasionally and unpredictably knock individual building blocks out of the structure of the genetic material,

provided, that is, that the entire cell does not die. The mechanism behind the idiokinetic effect of X-rays can be conceived in two ways: firstly, through the shower of electrons produced when X-rays and radium rays strike, and secondly, through the release of oxygen ions, which have an extremely destructive effect on all animal cells and on whose release, for example, the germicidal effect of iodine also appears to be based.

The possibility of idiokinetic damage caused by X-rays has recently been disputed by N ü r n b e r g e r, but only on the basis of entirely inadequate experiments and arguments. Of significance, however, is the following statement by Nürnberger: 'Despite all precautions and protective measures, it is impossible in a large X-ray facility to seal oneself off hermetically against the rays. This is already evidenced by the fact that even today many X-ray physicists and X-ray therapists are childless.' Of 30 married radiologists, 13 – that is, almost half – remained childless; the remaining 17 had an average of 1.2 children, totalling 21, of whom 19 were reportedly normal. And it is not even clear how many of the healthy children were born before their fathers began working with X-rays.

The idiokinetic effect of radioactive substances plays practically no role, as these substances are present only in very small quantities. X-rays, however, which can be generated in any quantity and are being used to an ever-greater extent for diagnostic and, in particular, therapeutic purposes, can have all the more disastrous effects, given that the doctors, technicians, assistants and nurses involved represent a select group of above-average competence.

Even in ordinary body cells, particularly those of the skin, X-rays can induce idiokinetic changes, namely the malignant degeneration of the cells. The nature of cancer lies in the fact that the idioplasm of a cell—which preserves its distinctive character and, amongst other things, regulates its growth—undergoes such a change that the cell begins to grow unchecked, destroying neighbouring cells.[F] Cancer now arises relatively frequently as a result of X-ray damage, and quite a few radiologists have already died of cancer. Cancer can also be caused by chemical substances; for example, there is a form of cancer known to affect chimney sweeps as a result of exposure to tar-like substances, as well as cancer affecting paraffin workers, bladder cancer affecting aniline workers, and lip cancer affecting pipe smokers. The cancerous degeneration associated with xeroderma pigmentosum (see p. 169) is due to an abnormally low resistance to the idiokinetic effects of sunlight. Even in healthy individuals, skin cancers develop almost exclusively on uncovered areas exposed to sunlight and chemical damage. Cancer can be compared to the so-called bud mutations in plants, which consist of individual cells undergoing idiovariations, from which modified shoots then emerge. Earlier, where hereditary predispositions to cancerous tumours were mentioned in certain contexts, their nature could not yet be clarified, as this requires an understanding of idiokinesis. Incidentally, X-rays or radium rays can not only cause cancer but, if conditions are favourable, can also bring about a cure, and this occurs as follows: cell nuclei that are in the process of division are particularly exposed to the effects of the rays, whilst the rays find far fewer points of attack in a resting nucleus. Since, in rapidly growing cancerous tumours, almost all cell nuclei are constantly dividing, whilst the nuclei of other body cells are usually at rest, it is possible to destroy all growing cancer cells with the rays, whilst the normal cells, with their resting nuclei, suffer only minimal damage. In principle, however, the triggering of cancerous degeneration and the destruction of cancer cells by radiation are merely two

different stages of a similar process. This, too, indicates that, when it comes to damage to germ cells, there are intermediate stages between complete destruction and complete integrity, in which changes occur in the idioplasm that are still reasonably compatible with life. Nürnberger, too, states that the only reason he probably did not obtain any diseased offspring from the irradiated animals in his experiments was that even small amounts of radiation cause the germ cells to die off. Fundamentally, he too cannot deny the idiokinetic effect of radiation, and the only conclusion that can be drawn from his experiments is that, given his limited number of cases, he did not succeed in demonstrating such an effect.

Idiokinetic changes apparently arise most easily during the maturation of germ cells, because at that stage their nuclei are not in a relatively protected state of rest but are engaged in complex processes of division. This is directly demonstrated by the aforementioned experiments by Towers. However, it is also supported by the fact that, under the influence of iodine and similarly acting poisons on the one hand, and X-rays on the other, germ cells during maturation and young fruits—in which almost all cells are constantly dividing—are most easily killed. Chemical abortifacients—such as some of the poisons mentioned above, but also others, for example Aloe and Juniperus sabina, which are frequently used by quacks—are also likely to cause idiokinetic damage on occasion, whether to the idioplasm of the foetus (in the event of a failed abortion attempt) or to that of the mother.

Syphilis is also frequently cited as a major cause of hereditary degeneration. It is certainly not impossible that metabolic products arising in the course of the disease may occasionally have an idiokinetic effect. For the most part, however, the similarity in the effects of syphilis and idiokinetic poisons is likely to be superficial. When children are born with syphilis to parents who have the disease, this is because the children themselves are infected with the syphilis pathogen whilst in the womb. It is, however, reported that children of syphilitic parents are often frail and sickly even when they themselves are free from actual syphilis. To explain this indisputable fact, however, it seems to me far more plausible to consider the idiokinetic effects of the poisons used as remedies against syphilis (mercury, iodine and arsenic compounds), which we cannot do without in treatment. In the treatment of syphilis, poorly soluble mercury salts are often injected into the muscles in fairly large quantities, where they remain for a long time and continuously release small amounts of mercury into the bloodstream, thereby inhibiting the growth of the syphilis pathogens. This can, of course, cause damage to offspring just as much as the inhalation of mercury vapour and dust in industrial settings. In the case of arsenic compounds (Salvarsan, etc.), damage to the genetic material is not equally likely, because arsenic is excreted much more rapidly.

The nature of idiovariations generally appears to depend less on the type of idiokinetic influences than on the previous constitution of the genetic material. Thus, in the experiments mentioned,

Towers exhibited quite distinct idiovariations under apparently identical external influences. Even in the extensive fly breeding experiments conducted by Morgan and his students, some idiovariations were observed on multiple occasions. Certain genetic populations are evidently particularly sensitive to idiokinetic influences. One can imagine that certain building blocks in the genetic material are particularly loose and can be dislodged more easily than others. Naturally, the loss of building blocks is also easier to achieve than the insertion of new ones. This is consistent with the observation that the majority of all idiovariations, the emergence of which has so far been tracked experimentally, are recessive relative to the parent form. In Morgan's breeding experiments, in which over 100 new idiovariations have already been observed, around four-fifths were recessive, and were therefore probably due to defects in the genetic material. It must also be borne in mind that dominant forms are much easier and more reliably identified than recessive ones; in reality, therefore, the number of recessive forms is likely to be even greater. It is also highly noteworthy that the vast majority of Morgan's new idiovariations had a lower capacity for adaptation than the parent form; they are therefore to be regarded as pathological. Only in a relatively small number of cases was a reduction in the probability of survival not immediately apparent. The same applies to the idiovariations observed by Baur and other researchers. Incidentally, pathogenicity need not always be expressed in external appearance. Several idiovariations in Morgan's breeding lines manifested themselves solely in the fact that their carriers died more or less suddenly ('lethal factors').

Since the carriers of pathological idiovariations, which cause a more severe condition, often do not reproduce, the inheritance of the predisposition cannot, of course, be observed in these cases. This may explain some of those observations where, in an otherwise healthy family, there is only a single case of a condition otherwise known to be hereditary. It has already been shown that such isolated cases are to be expected with recessive disorders simply as a manifestation of recessive inheritance. Moreover, however, one must bear in mind that even new idiovariations, which would in themselves be dominant, remain isolated in all cases where they do not result in reproduction.

It is therefore by no means the most severe idiokinetic disturbances of the genetic material that are of the greatest significance for the life of the race. Where the effect is very severe, the germ cells die off; the disturbance of the genetic material cannot therefore be passed on. In cases of lesser damage, the foetus often dies in the womb, so that even in this instance the spread of pathological idiovariations is out of the question. In cases of even lesser damage, newborn children often die from congenital weakness, and as the degree of damage decreases further, children die before reaching reproductive age. It is precisely relatively minor damage to the genetic material—which is compatible with life to such an extent that those carrying it can more or less hold their own in life and reproduce—that therefore poses the greatest threat to the vitality of the race.

Of course, the causes of new pathological hereditary traits are by no means limited to the toxins and other gross forms of damage mentioned above. When wild animals are subjected to the conditions of artificial breeding, this alone seems to be sufficient to give rise to pathological hereditary traits in large numbers (cf. p. 116). If, for example, a species of butterfly is bred in captivity, such severe degeneration regularly occurs after just a few generations that further breeding becomes very difficult or is no longer possible at all, as I have observed in numerous breeding programmes. Weismann observed the same phenomenon in the breeding of field mice. But even in the wild, idiovariations occur time and again in no small number. Idiokinetic influences are therefore at work there too.

> The effects of sunlight, particularly ultraviolet radiation, undoubtedly also give rise to electron radiation in the wild on many occasions, although naturally not nearly as concentrated as in an X-ray laboratory. But occasionally, even in the wild, free-flying electrons may well trigger changes in the genetic material. Furthermore, atomic decay—which gives rise to radium radiation—is not limited solely to highly radioactive substances; it also occurs, to a lesser extent, in other substances.

Since most of the idiovariations that have been observed are recessive, it is generally not to be expected that an idiokinetic damage affecting a person's genetic material will manifest itself in their children. Rather, it is to be expected that the genetic material of the other parent does not contain exactly the same defect, and

that the defect therefore remains masked for the time being. This may be illustrated by a diagram.

Fig. 66.
Diagram showing the first manifestation of a recessive pathological predisposition.

Suppose that a particular genetic trait in the germ cell of a drinker is destroyed by alcohol. Then a child of the drinker will carry a corresponding recessive pathological predisposition in a masked form (represented by a dot within a circle). Since the drinker's child will, as a rule, marry a partner who does not carry the same defect in their genetic material, the pathological predisposition will not yet manifest itself in the grandchildren either. However, half of the grandchildren will carry the allele in a masked state. As the carriers of the pathological allele in the grandchild generation are all siblings, they cannot be considered as potential partners for having children; consequently, the allele will not manifest itself in the great-grandchild generation either. However, half of the grandchildren who are carriers of the allele will, in turn, have children who are also carriers of the allele. In the great-grandchildren's generation, therefore, siblings may carry the trait in a recessive state; and in the great-great-grandchildren's generation, marriages between cousins in the great-grandchildren's generation may therefore produce children who are now actually afflicted with the condition, the predisposition to which had already arisen in the genetic material of their great-great-grandfather.

If a recessive hereditary condition appears for the first time in a population today, it must therefore be assumed that the pathological genetic predisposition actually arose more than 100 years ago, perhaps around the time of the Napoleonic Wars. And that would in fact be the earliest possible time of manifestation. Since marriages between first cousins are not the norm, recessive hereditary disorders observed today may therefore have originated as far back as the Thirty Years' War or even earlier.

Perhaps this explains the peculiar clustering of cases of spinocerebellar ataxia in a family group studied by Rütimeyer and Frey. In the second half of the 19th century, in a small Swiss village whose population essentially intermarried amongst themselves, a total of 15 cases of spinocerebellar ataxia (cf. p. 220) occurred across six different families. As is typical with recessive inheritance, the parents of the affected individuals were also free from the condition in this case. Nor are there any known cases of the condition in earlier generations. All six families in which affected members occurred could be traced back to a common ancestor named *Glaser*, who was born in 1510. It may therefore be that the recessive genetic predisposition to ataxia had already arisen at that time, but that the condition only manifested itself in various families after 10 to 11 generations, following the convergence of two such predispositions as a result of inbreeding within the established population. In any case, the suggestion that the genetic makeup of these families had been undergoing a gradual process of degeneration since the time of that common ancestor, which then ultimately led to the sudden onset of similar severe cases of the disease in the 10th or 11th generation, is entirely unacceptable. On the other hand, it certainly seems possible to me that the recessive trait had been present in the population for even longer. It is difficult to rule out with certainty that cases of ataxia may not also have occurred there in the 18th or 17th century. Furthermore, attributing the condition to the aforementioned progenitor *Glaser* in the line of descent is more or less arbitrary

or coincidental. In a small, remote village, most families are likely to have not just one, but a whole number of common ancestors in the 10th or 11th generation.

Dominant genetic variations naturally manifest themselves in children. Among recessive variations, sex-linked ones occupy a special position. As males possess only one sex chromosome, defects in the genetic material of the sex chromosome can be masked in females but not in males. This is likely to explain why, among miscarriages, male foetuses are significantly more numerous; as far as can be ascertained, the ratio is at least 150 to 160 : 100. Mortality rates among boys are also higher in infancy and childhood. Damage to the other chromosomes, on the other hand, is initially masked in males as well.

In some cases, a change in the genetic material presumably arises simply from a disruption of the antagonism between homologous genetic units. As explained in the general section, two homologous genetic units generally always separate during germ cell formation. However, there is much to suggest that this rule is not without exception. For instance, Bridges was able to demonstrate that, in exceptional cases, two homologous chromosomes may enter the same germ cell or both may be absent from it. The same is likely to occur with the segments of the chromosomes, the chromomeres. If, during reduction division, the chromomere chain (see p. 48) in two homologous chromosomes fails to break at exactly the same point, the result will be that a chromomere ends up on the 'wrong' side. In this way, germ cells will arise which do not contain the chromomer in question at all, and others which contain it twice. Such a loss of a chromomer or a unit of heredity will, furthermore, have exactly the same consequences as the destruction of a unit of heredity by chemical or physical influences; that is to say, a recessive defect will henceforth exist in the genetic material. It is even not unlikely that the majority of all loss variations arise from such a disruption of the antagonism between genetic units. If the maturation of germ cells takes place under the disruptive influence of idiokinetic factors, such as narcotic substances like alcohol, the smooth exchange of chromomers will naturally be particularly susceptible to disruption.

Such a disruption of the antagonism would, of course, lead just as often to the duplication as to the loss of genetic units within a genetic mass, and it may be surmised that this is how the occurrence of polymeric factors—that is, several identical genetic units within a genetic mass—may first come about.

The easiest to understand are those genetic changes resulting from the simple loss of a single genetic unit, followed by those based on the accumulation of similar genetic units. Apart from these more quantitative changes, however, there must of course also be what are essentially qualitative changes, arising from additions and rearrangements within the genetic units.

Of course, idiokinesis does not merely give rise to new pathological genetic traits; rather, all those countless genetic changes which have enabled the development of living organisms in the course of their phylogenetic history are

thought to have arisen idiokinetically. However, this can only occur in conjunction with rigorous natural selection, which results in the preservation of the few idiovariations that confer enhanced adaptive capabilities. Under our present conditions, where such rigorous selection is absent, idiokinesis has, for the most part, a wholly deleterious effect. Since the loss of genetic material is far more common than the emergence of new genetic material, and since most dominant idiovariations by no means represent an increase in adaptive capacity, idiokinesis leads to a progressive deterioration of the genetic pool and to progressive degeneration. Furthermore, selection can also contribute to an increase in degeneration, namely in the form of so-called counter-selection, as will be shown in the second volume.

Whilst the term 'disease' refers to both more or less temporary impairments of adaptive capacity and permanent ones, we restrict the term 'degeneration' to hereditary traits. A population may therefore be heavily affected by non-hereditary diseases, such as syphilis, yet it need not for that reason be degenerate. We generally understand degeneration to mean the emergence and spread of pathological hereditary traits, that is, the loss of the hereditary fitness of the ancestors. Degeneration is the central problem of racial hygiene; the prevention and combating of degeneration is its essential aim.

Section Four.

The Heredity of Intellectual Ability.

By **Dr Fritz**

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1. Outstanding talent.

Even in our discussion of pathological hereditary traits, we have encountered certain facts which allow us to draw conclusions regarding the heritability of normal mental abilities. For instance, on p. 165 it was pointed out that we can infer from the inheritance of colour-blindness that of normal colour vision—that is, a mental ability. Similarly, one can infer the heritability of normal hearing from the occurrence of recessive genetic traits that cause deaf-mutism. In a population in which the vast majority of people were deaf-mute, normal hearing could be regarded as a dominantly inherited trait; indeed, it would appear to be a peculiar, superior ability to perceive events without seeing them—an ability of which the majority of the population could have no real conception. From the occurrence of hereditary mental deficiency, we can conclusively infer the hereditary nature of normal intellectual faculties; and from the fact that there is a whole range of different types of hereditary mental deficiency, it further follows that a whole number of hereditary units are involved in the development of normal intellect, none of which may be missing without defects of intellect becoming apparent. In a population consisting entirely of people with mental deficiency, normal ability would stand out as a special hereditary capacity of the mind, which, however, the vast majority of the population would be unable to comprehend.

The situation is quite similar with regard to the heritability of abilities that exceed the average of today's population to a degree comparable to that by which the abilities of the mentally deficient fall short of the average. In German, such outstanding abilities are usually referred to by the two loanwords 'Talent' and 'Genie', whereby 'Talent' tends to denote more one-sided abilities, particularly in the artistic sphere, whilst 'Genie' denotes versatile and comprehensive abilities. On the other hand, of course, one also has in mind a difference in degree. Just as, for example, idiocy represents a higher degree of mental deficiency compared with mental retardation, the term 'genius' is intended to denote a particularly high degree of outstanding ability compared with mere 'talent'. It is imagined that, in a population of one million, whilst there may be many 'talents', there are at most very few
'geniuses'.

Whilst it is understandably difficult to trace the ordinary differences in aptitude in terms of their heritability, this is considerably easier in the case of outstanding aptitudes, simply because they stand out more clearly from the average. Particularly striking, for example, is the occurrence of musical talent within families. In the *Bach* family, a high level of musical talent can be traced in an unbroken line through five generations of the male line; among *Johann Sebastian Bach's* sons were no fewer than five significant musicians, and several other outstanding musicians also emerged from the descendants of *Bach's* great-grandfather. *Beethoven's* father and

grandfather were highly gifted musicians. Similarly, musical talent can be traced through three generations in the *Mozart* family. In the case of many other great musicians, the hereditary nature of their talent is presumably not so clearly evident simply because the predisposition was naturally often inherited through the female line, where it did not have the same favourable opportunity to develop. Thus, the composer *Mendelssohn* seems to have inherited his talent mainly from his mother's side.

Musical aptitude seems to depend essentially on the presence of very few, or at most a few, genetic units. This is not, of course, to be understood as meaning that the musician might lack the other genetic units which determine the physical and mental constitution of a normal person; however, these other genetic units—which must be present in the musician in addition to the strictly musical aptitudes—are evidently otherwise quite widespread; and therefore the strictly musical aptitude may appear to be inherited in a dominant manner. A distinctly musical sensibility is not at all uncommon even among the mentally disabled; naturally, however, a mentally disabled person cannot become a great musician. Creative activity in the field of music, in particular, requires not only an exceptional musical ear, but also a rich emotional life, a lively imagination and intellectual creativity. This is evidently linked to the fact that, whilst one usually finds other talented musicians in the family of a musical
'genius' there are usually other talented musicians, but not a second genius. We need not, therefore, assume that *Johann Sebastian Bach* surpassed his musical relatives specifically in terms of his musical aptitude; rather, it suffices that this was combined with particularly favourable intellectual aptitudes in other areas. The distinction between the one-sided gift of talent and the comprehensive gift of genius is likely to arise in a similar manner in other fields as well.

Much like musical aptitude, mathematical aptitude also occurs in a one-sided form, that is to say, without necessarily being linked to other high levels of aptitude. The *Bernoulli* family of mathematicians has become particularly famous, having produced no fewer than 8 (or, by a less strict count, even 9) eminent mathematicians. It is highly significant

that several of them had initially turned to other professions; one had embarked on a career in commerce, two had begun studying law and one had begun studying biology; yet, despite this, three of these four eventually became professors of mathematics and one of physics.

Outstanding talent for painting and sculpture also runs distinctly in families. For instance, nine painters emerged from *Titian's* relatives. In the visual arts, even more so than in music, it is the case that, in addition to the actual artistic talent, various other intellectual aptitudes must be present for significant achievements to emerge. Great visual artists therefore generally come from families from which significant figures in other fields also emerge. This applies, for example, to the family *of Anselm Feuerbach.*

In the *Krupp* family, a talent for technical inventions can be traced back through three generations. In the *Siemens* family, three (or, according to some accounts, four) brothers out of fourteen siblings were notable inventors.

Galton himself had already described a whole series of outstanding families, and many others have since come to light. *Galton* was *Darwin's* cousin, and he was scarcely less intellectually significant than Darwin; the fact that he did not make a discovery of equal world-shaking significance does not, of course, imply that he was any less gifted. Nevertheless, *Galton* became the founder of modern eugenics. In the field of genetics, he even arrived at far clearer concepts than his cousin *Darwin*; he can indeed be regarded, alongside *Mendel,* as a pioneer of modern genetic research. Apart from *Darwin* and *Galton*, a whole host of other eminent figures have emerged from the same family circle; their common grandfather, *Erasmus Darwin,* grasped the fundamental idea of the theory of evolution even earlier than Lamarck. *Charles Darwin's* marriage to a cousin also produced three eminent researchers and scholars.

H. W. Rath in Württemberg has traced a very interesting family circle. The great poets *Hölderlin*, *Uhland* and *Mörike*, the famous philosophers *Schelling* and *Hegel*, the poet and aesthetician *F. Th. Vischer*, the storytellers *Hauff*, *Schwab* and *Ottilie Wildermuth*, and a whole host of other renowned artists and

scholars all share a common ancestor named *Bardili*, who was a professor of medicine in Tübingen at the end of the 16th century and was married to the daughter of *Burckhardt*, a professor of logic. According to Rath, the vast majority of famous Swabians are descended from this couple.

In such cases, of course, it cannot simply be a matter of a dominant trait spreading through a family circle, such as having six fingers or night blindness. Rather, the concentration of exceptional talent in such family circles is evidently due, to a large extent, to the fact that distinguished families have a preference for marrying amongst themselves.

Galton also carried out a statistical analysis of the family relationships of around 1,000 of England's most famous men. He found that
100 outstanding men had, on average, 31 distinguished fathers, 41 distinguished brothers, 48 distinguished sons, 17 distinguished grandfathers and 14 distinguished grandsons. These figures only come into their own when one considers that Galton defined the concept of outstanding talent so narrowly that there are only about 250 outstanding men in a million, or one in 4,000.

In America, F. A. Woods traced the family relationships of 3,500 well-known Americans. Whilst any American citizen had a 1/500 probability of being closely related to one of them, the probability of one of these well-known men being related to another was 1/5 – that is, a hundred times greater.

> The heritability of intellectual ability is often disputed on the grounds that 'geniuses' do not in turn have sons who are equally gifted. Whilst this is undeniably true, it naturally proves absolutely nothing against the hereditary nature of intellectual ability. We have seen above that a whole host of hereditary factors must coincide for an outstanding and comprehensive talent to emerge. However, each child inherits only half of its genetic endowments from each parent, and as a rule, this is naturally not exactly the better half of those endowments. Even if, therefore, the wives of geniuses were to regularly possess the same favourable genetic endowments as their husbands—which, of course, is almost never the case in reality—it would still not be to be expected that the sons of geniuses would equal their fathers. Like all mixed variations, genius is not hereditary as such in mixed populations; nevertheless, the individual traits of which it is composed are hereditary. With appropriate selection and pure breeding, however, genius would be hereditary just like other mixed variations. One could therefore also 'breed' 'genius'.
>
> In earlier times, when significant differences were observed between father and son or between siblings, these were seen as evidence against the significance of

heredity. Since the discovery of Mendel's laws, however, we have come to realise that such differences— —may well be attributable to the effects of the laws of heredity. Mendel's laws explain both the fact that highly musical siblings may coexist with those who are completely unmusical, and the fact that the sons of brilliant men do not tend to resemble their fathers.

In most areas of intellectual life, a whole range of favourable aptitudes must come together for outstanding achievements to emerge: strong powers of judgement, the ability to distinguish concepts clearly, a gift for synthesis, a sense of truth, steadfastness of will, quick comprehension and a good memory. Depending on how pronounced these various aptitudes are, the degree of talent naturally varies. The scholar requires more receptive aptitudes, such as quick comprehension and a good memory. There are families in which several or all members possess a remarkably good memory; such families often produce scholars, particularly philologists, historians and theologians. A good memory usually goes hand in hand with a keen interest in the unique and the particular – in short, in history – whilst a sense of the general and the universal tends to stand in a certain contrast to this. Yet it is precisely this sense that the researcher and inventor require, alongside strong powers of judgement and the ability to intuitively grasp universal relationships. The philosopher also needs an incorruptible sense of truth that does not shy away from painful truths. The entrepreneur, the statesman and the military commander must be endowed with a keen insight into human nature, a strong will to exercise power and great foresight. The creative artist and the poet must possess a vivid imagination and creative power, as well as the ability to empathise with the souls of other people. Good physical health is highly conducive to outstanding achievements; without it, sustained intellectual work is scarcely possible. Essential, however, is a keen interest in a cause; in most cases, this interest is made even more effective by a lively drive to distinguish oneself, generated by a strong intellectual or material will to power.

'Ambition' or 'vanity' are almost invariably viewed with a certain disapproval in the case of great men; yet there can be no doubt that the disposition denoted by these terms—for which we prefer to use the morally neutral term 'need for recognition'—is one of the most essential driving forces behind great achievements, even if often only unconsciously. One might be inclined to object that this, then, is not 'true' greatness. Well, greatness in this sense is a value judgement that is, strictly speaking, not at all comprehensible in scientific terms. What we can actually ascertain is merely fame. Success in the world and fame, however, naturally depend not only on objective achievement, but above all on a charismatic demeanour, an instinctive sense of what makes an impression, and a skill for written expression or eloquence – abilities which, for example, *Goethe* possessed to an outstanding degree. On impartial reflection, the powerful impact on contemporaries and posterity must also be recognised as a form of ' '. In times past, the man who was physically stronger than all others (Siegfried) was regarded as the greatest; today, it is the man who captivates people's souls.

2. The usual differences in aptitude.

Naturally, the heritability of ordinary talents is far less striking than that of exceptionally high talent. Nevertheless, this too can be established not only through everyday experience but also using scientific methods.

Professor Peters, a psychologist at the University of Würzburg, collected the school reports of 1,162 children and compared them with those of their parents and grandparents. The assessment of a child throughout their school career and by different teachers generally remained largely consistent, meaning that conclusions drawn from the average values can certainly be considered reliable. On average, the children's school reports deviated from the mean in the same direction as those of their parents; indeed, the children's deviation amounted on average to about one-third of that of their parents. This phenomenon, which Galton termed 'regression', does not, incidentally, suggest that heredity plays a relatively minor role, for even if every single predisposition were strictly hereditary, one would hardly expect a lesser
'regression' be expected. After all, each child inherits only half of their parents' genetic endowments, and these are combined differently in the children than in the parents. If the parents are now grouped according to their academic performance, the best group naturally comprises the particularly favourable combinations, whilst the worst group comprises the particularly unfavourable ones. And since, in children, the paternal and maternal traits combine to form new combinations, it is by no means to be expected—even in the case of strict heritability—that the children of parents grouped in this way would again deviate so strongly from the average. Rather, this would only be expected in the hypothetical case where both parents were always completely identical in their idiotypic constitution and, moreover, consistently homozygous. However, since the population is in reality highly heterozygous and parents are never completely identical, the deviation in the same direction amounting to one third already indicates that heredity plays a far more significant role than external influences.

When comparing the average marks of the grandparents with those of their grandchildren, a correlation of only slightly lesser magnitude was found. Peters went on to compare the school reports of children whose parents had the same average marks separately with those of the grandparents

and found that, even in these cases, the children deviate from the mean in the same direction as their grandparents. Since the effect of a shared environment cannot account for this similarity, it must be regarded as particularly strong evidence for the heritability of aptitude. On the basis of his research, Peters even concludes that the environment (upbringing at home, etc.) has only a minor influence on academic performance.

If the academic performance of both parents differed, the performance of the children of the same pair of parents also differed more markedly on average from one another than when the performance of both parents was approximately the same. Peters rightly sees this as an expression of Mendelian segregation.

Schuster and Elderton have also statistically investigated the psychological similarity between siblings among schoolchildren and found that it is almost exactly as great as the physical similarity. When comparing the school reports of fathers and sons, they found a similarity almost as great as that in physical characteristics.

Similarly, Thornike was able to establish a significant similarity between siblings in terms of academic performance. He also compared 50 pairs of twins and found that the similarity between them was more than twice as great as is usually the case among siblings. This is evidently due to the fact that the sample included a number of monozygotic twins, who, of course, share an identical genetic endowment, whereas dizygotic twins are no more similar to one another than siblings generally are (cf. pp. 86 and 187). The striking psychological similarity between identical twins also reveals the paramount importance of hereditary factors in shaping human mental faculties.

A comprehensive study by Heyman and Wiersma has also provided striking evidence of the hereditary nature of psychological predisposition. On the basis of a questionnaire survey of 437 families with 1,541 children, the heritability of 181 out of 185 traits enquired about could be traced.

> Sex-linked genetic factors are undoubtedly also of great significance for intellectual ability. In our earlier discussion of pathological genetic factors, we encountered several diseases of the central nervous system that are sex-linked recessive, and which therefore stem from defects in the sex chromosome (cf. e.g. p. 219). It follows from this

it inevitably follows, however, that there must normally be genetic factors in the sex chromosome which are indispensable for the development of a normal central nervous system, and we may further conclude from this that at least some of these sex-linked genetic factors will also manifest themselves in the psychological makeup of their carriers.

Clear evidence of sex-linked inheritance can be found in the studies cited by the psychologist P e t e r s. When he did not simply compare the average of the parents with that of the children, but analysed the data separately by sex, the following became apparent: The relatively lowest degree of similarity was found between fathers and sons, whilst the highest was between mothers and daughters; indeed, the latter was over 70 per cent greater than the former. The similarity between fathers and daughters and that between mothers and sons fell in the middle; the former was about 12 per cent greater, and the latter about 30 per cent greater than that between fathers and sons. Certain mathematically defined coefficients, the detailed discussion of which would take us too far afield here, were used as a measure of average similarity (see p. 249). Two different causes may explain these deviations from the general average similarity between parents and children: firstly, the sameness or difference in sex, and secondly, sex-linked genetic factors. The relatively high degree of similarity between mothers and daughters can be explained by the fact that, in this case, sex-linked genetic factors, as well as the fact that they share the same sex, reinforce the general similarity between parents and children, so that this is exceeded by more than 20 per cent. In every case, a mother and daughter share one of their two sex chromosomes, in which the sex-linked genetic traits are located. A father and son, on the other hand, never share a sex chromosome; and so it is that, despite sharing the same sex, their average similarity lags behind the general average similarity between parents and children by almost 30 per cent. In this case, therefore, the differences in sex-linked genetic traits prove to be more significant than the fact that they share the same sex. This is also reflected in the fact that the similarity between father and daughter, despite the difference in sex, was still found to be 12 per cent greater than that between father and son, because in the former case there is a shared sex chromosome, whereas in the latter there is not. These circumstances are likely also to be linked to the fact that Peters found a considerably greater psychological similarity amongst sisters than amongst brothers. Sisters always share at least one sex chromosome, namely the one inherited from their father, and in half of the cases even two. Amongst brothers, on the other hand, the difference in their single sex chromosome is just as common as its similarity.

These findings can be regarded all the more as evidence for sex-linked inheritance of psychological traits, given that Peters himself was apparently entirely unaware of the theory of sex-linked inheritance whilst conducting his work, and thus proceeded entirely without bias.

The findings of Heyman and Wiersma suggest sex-linked inheritance, as evidenced by a greater average similarity between mothers and children compared with that between fathers and children. The father possesses only one sex chromosome, whereas the mother has two, through which sex-linked genetic traits can be passed on to the children. Furthermore, sex-linked recessive traits that manifest in the father generally do not manifest at all in the children, which also contributes to a comparatively lower degree of similarity between fathers and children.

In the aforementioned studies by Schuster and Elderton, a sign of sex-linked inheritance can probably be seen in the fact that the psychological similarity between brothers was found to be approximately 33 per cent greater than that between fathers and sons. Brothers

do, after all, share a sex chromosome in half of all cases, whereas fathers and sons never do.

The phenomena of sex-linked inheritance may also explain the common belief that sons usually take more after their mothers, whilst daughters take more after their fathers. However, it is also evident from the circumstances outlined that this belief represents only a very superficial approximation of the true state of affairs.

The same applies to a theory which Schopenhauer formulated and tenaciously defended long before the advent of scientific research into heredity, namely that 'intellect' is always inherited solely from the mother, whilst 'character' is inherited exclusively from the father. There can be no doubt whatsoever today that 'intelligence' does not constitute an idiotypical unit that is inherited as such; rather, it is built up from a large number of hereditary factors; and the same applies to character. Nevertheless, Schopenhauer deserves some credit for having pointed out, even at that time, the greater significance of the mother for the intellectual aptitude of her sons, and for having provided a number of historical examples to support this.

However, only a fraction of all psychological dispositions can ever be sex-linked. An unfertilised human egg cell contains 12 chromosomes, including one sex chromosome. One might therefore expect that approximately one-twelfth of all human genetic factors will be inherited in a sex-linked manner. A review of pathological genetic traits suggests that this estimate is likely to be roughly accurate. However, a larger proportion of precisely those genetic traits that are of essential importance for psychological predisposition is likely to be localised in the sex chromosome.

Finally, it should be briefly noted that the psychological difference between the sexes is itself idiotypical, that is to say, hereditary. One cannot say that women are less gifted than men; they are simply gifted in a different way. In terms of perception and memory, women are on a par with men; in conceptualisation and independent judgement, however, they are not, on average. Of course, there are also many women who are superior to the majority of men in these respects; on average, however, the situation is different. Great women, in the sense of possessing outstanding creative talent, have scarcely made a name for themselves. Even more pronounced than the differences in intellectual aptitude are, apparently, the differences in the instinctual life of the two sexes, not only in sexual instincts but also in nutritional instincts and, in particular, in social instincts. The ability to control one's desires is normally greater in women than in men. The need for recognition is directed towards other things. It is simply that women have quite different tasks to fulfil in the life of the race, but these are no less important than those of men; and if one of the two sexes were dispensable, it would be the male and not the female.

Almost as important as the realisation that psychological dispositions are hereditary is the insight that everything a person acquires in life through practice and experience is not inherited. A person with a certain musical aptitude may learn to play the piano or take up some other musical activity; yet the musical talent of their offspring will be neither greater nor lesser as a result than it would have been without it. What a human being or any other living creature inherits is a certain set of potential responses (cf. p. 16). Which of these potential responses are realised and how

they are realised is, however, of no significance for the predisposition of their offspring. Where a potential response is entirely absent, it cannot, of course, be acquired. There was a time when people tried to make deaf people hear by producing loud noises beside them. That seems at least a little ridiculous to us today. No amount of auditory exercises or punishment will make a deaf person hear. A colour-blind person cannot, through any amount of practice, acquire the colour perceptions they lack; rather, the nature and degree of their colour blindness always remain the same. For success to be achieved through practice and education, the appropriate predispositions must already be present; then, within certain limits, development is possible. The successes of education rest chiefly on the acquisition of knowledge stored in the memory and on becoming accustomed to certain views and modes of behaviour. Both memory and mental malleability in general are at their greatest in youth; this is the time when a person adopts knowledge, views and habits that have, to some extent, stood the test of time in the society to which they belong. What is absorbed in youth becomes 'second nature', but 'first nature', which is rooted in one's genetic makeup, remains unaffected by it. The educational efforts of past generations therefore have no influence on the predispositions of the present generation, nor do those of the present generation have any influence on the predispositions of future generations. The mental assets inherited from one's forebears need not first be acquired by one's children, and everything that one's forebears have acquired on the basis of their hereditary predispositions must also first be acquired by their descendants in order for them to possess it.

Experimental evidence clearly refutes the possibility of the 'inheritance of acquired characteristics'; theoretically, we would be unable to conceive of such a thing; nor does human life offer any experiences that would have to be interpreted in this way. If the children of educated parents are, on average, more gifted than those of uneducated parents, this is not due to their parents' education, but because they have inherited traits from them that already enabled their parents to acquire an education. It is therefore the capacity for education that is hereditary. On the other hand, it is a common experience that educated families often produce sons who lack talent and who, despite all the effort expended on educational resources, are unable to

acquire a higher level of education. Such experiences clearly contradict the notion that educational success is hereditary, whilst they are readily understandable on the basis of Mendel's fundamental law of heredity. It is therefore entirely hopeless to achieve upbringing and practice to permanently elevate the human race. This is one of the most important insights in the field of racial hygiene.

Anyone who finds it disheartening that everything we might achieve through our ceaseless endeavours to perfect ourselves cannot, after all, be passed on to the genetic heritage of our descendants should be reminded that, were it otherwise, future generations would also be burdened with all the chaos of error and nonsense, of contemptibility and baseness, from the past and present. Insofar as these do not stem from the hereditary nature of our contemporaries, but only from the circumstances of the times, our descendants need not be burdened by them. Future generations can work their way back up to greatness and purity, provided we ensure that they are descended from a capable ancestral heritage. That is what it all comes down to. It is therefore the great question of destiny for our race and culture whether the capacity for judgement of our contemporaries is still sufficient to counter the prevailing prejudices regarding the omnipotence of the environment and to bring about the necessary transformation of our outlook on life and all our institutions, or not.

3. Talent and psychopathy.

Significant deviations from the average are usually pathological; though by no means in every case, as was already emphasised on p. 144, but certainly in all those cases where the nature of the deviation results in a reduced probability of survival compared with the average. This is often precisely the case with outstanding talents. Numerous talented people fail to cope with the demands of everyday life, which average people of sound mind meet without difficulty. What applies to personal self-preservation applies to

even greater measure to the preservation of the family. A large proportion of talented people—and all the more so of those of genius—remain unmarried; others live in unhappy family circumstances, because the genius's ability to adapt often fails even in relation to their spouse. Almost invariably, this leads to the rapid extinction of the family. However, when a person's mental constitution impairs the preservation of the family, we speak of psychopathy and, in the most severe cases, of mental illness. We must therefore not shy away from discussing the question of whether genius is a pathological condition, especially as it is of considerable significance for the practical stance of eugenics towards psychopaths.

In order to discuss this question without prejudice, it is necessary to draw a clear distinction between the scientific and the value-judgemental perspectives. Illness is a scientific concept, and it need not necessarily be burdened with a negative value judgement in all circumstances. Perhaps certain pathological conditions may even be desirable; but that is no reason to deny that they are pathological in nature. The identification of a pathological mental state in a genius therefore does not diminish the value of their achievements. Illness is not synonymous with inferiority. On the other hand, however, the co-occurrence of outstanding achievements and pathological traits of mental life is far too frequent to be explained by mere chance; and since pathological mental states are generally hereditary, the significance of pathological predispositions in this regard must not be overlooked when discussing the hereditary basis of intellectual giftedness.

> The catchphrase 'genius and madness', which is often used in the wake of Lombroso—who was the first to discuss this question at length—is misleading, because in cases of actual mental illness, particularly those accompanied by damage to the brain, brilliant creations are naturally not to be expected. Where such conditions have been observed in brilliant individuals (e.g. *Hölderlin*, *Scheffel*, *Schumann*, *Nietzsche*, *Maupassant* and others), they have evidently impeded rather than fostered their achievements. Nevertheless, it is conceivable that, on occasion, such illnesses may contribute to the emergence of exceptional achievements by breaking down normal inhibitions.
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> Epilepsy, too, is certainly not conducive to genius in general. I would therefore venture to suggest that in the case of those great men of whom epilepsy is reported (e.g. *Caesar*, *Paul*, *Muhammad*, *Francis of Assisi*, *Alfred the Great*, *Napoleon I*), this was either merely a coincidence or, more likely, epilepsy-like manifestations of orgorgism, which in earlier times were apparently frequently conflated with epilepsy as a 'holy disease'.

Orgorism (see p. 234) undoubtedly plays a major role in the attainment of fame. *Rousseau*, *Frederick the Great*, *Wagner* and *Nietzsche* (apart from his later mental illness), for example, display orgoristic traits. One could name numerous other figures of equal stature; indeed, it even seems to me that this applies to the majority of famous men, at least in artistic, political and related fields, whilst it does not apply to the same extent to the great researchers and explorers. The abnormal susceptibility to desire can make goals appear attainable to the Orgorist that a sober mind would regard as hopeless from the outset. 'I do not know the word "impossible"', said *Napoleon*. Orgorism can create a faith that moves mountains, a faith that can defy all evidence of the senses and reason, and which, precisely for that reason, occasionally accomplishes the extraordinary.

'Whoever approaches the gates of poetry without the madness of the Muses will see their poetry pale in comparison to the art of those who possess creative madness.' So said Plato, and Aristotle, too, had already pointed to the pathological disposition of the great poets and artists, philosophers and politicians. Ibsen has, for the most part, brought only orgorists to the stage as significant figures; and they come across as quite true to life there; his *Peer Gynt*, who represents a caricature of an orgorist, has enjoyed great success throughout the world. In general, 'orgorism' seems to favour success with people more than actual intellectual achievement. There is something captivating about the belief in dazzling idealised images; and an extraordinarily vivid need for recognition drives many 'orgorists' time and again to words and deeds that promise to make an impression on their contemporaries and posterity. Just as orgorism can lead to the mimicry of illness and weakness (hysteria), so too can it give rise to a mimicry of intellectual greatness; and this is scarcely less common or impressive than the former. In individual cases, the predisposition may, of course, be associated with a wide variety of other traits; and without outstanding talent in other areas, orgorism naturally cannot produce significant achievements either.

Psychasthenia ('neurasthenia'), which is related to orgoristia, also appears in some cases to be conducive to brilliant achievements. *Darwin* was a psychasthenic who could work for only a few hours a day and yet achieved more than a thousand other researchers. The same applies to *Fechner*.

The well-known psychiatrist A s c h a f f e n b u r g even states: 'Nervousness and genius are two manifestations of the same psychopathic predisposition. The very same sensitivity and receptivity that make the neurasthenic's nervous life appear pathological give him the capacity for unexpected and bold combinations, make his thinking and feeling deeper and more enduring, and endow him with the vigour of the artist and poet, the dynamism of the genius.' According to the classification adopted in the chapter on mental anomalies, however, some of these traits would in fact be better classified as orgorism.

> The genius's brain is almost constantly in lively activity, and whilst it may show signs of exhaustion more frequently than the average brain, this need not necessarily be due to abnormal fatigability, which has been described as characteristic of psychasthenia. An average brain might fail after only a fraction of the effort expended by a genius's brain, and it may be spared the signs of psychasthenia simply because it does not, as a rule, exert even that fraction of effort; conversely, a considerable proportion of what appears in ordinary life as neurasthenia may be due to excessively vigorous mental activity. And if the abnormally intense activity of the genius's brain were, in itself, to impair health, then it would, as such, already be pathological.

The parathymic (manic-melancholic) disposition also seems capable of contributing to genius. *Konrad Ferdinand Meyer* and *Anselm Feuerbach* suffered from mental disorders of this type. According to the usual classification, one would also include the illnesses *of Robert Mayer*, the discoverer of the law of energy, and *Gregor Mendel*, the discoverer of the fundamental law of heredity, in this category. *Schopenhauer* had a melancholic disposition and at the same time displayed orgoristic traits. Möbius also attributes a parathymic disposition to *Goethe*; yet it seems to me that the pathological signs in his case point more towards orgorism, which can manifest itself in a manner very similar to mild parathymia.

A paranoid disposition can evidently also be of undoubted significance for outstanding achievements. The pathological tenacity with which the paranoid individual defends his convictions can enable him to achieve feats that other people of otherwise equal talent are unable to accomplish. The well-known social hygienist Grotjahn tends to regard almost all tenacious pioneers of a movement or an idea as mild paranoids. According to my observations, orgoristia is likely to be of greater significance. Perhaps, however, there are close connections between the orgoristic, paranoid and parathymic predispositions (cf. p. 236).

> In any case, it is entirely mistaken to believe that no sound insights or beautiful works of art can spring from a diseased mind. What is objectively true and false is often far too difficult to determine for this distinction to be taken as a hallmark of mental illness. False beliefs can occasionally be life-affirming, whilst true ones may be contrary to life. The falsity of a thought therefore proves nothing in favour of, nor does its truth prove anything against, the mental illness of its author. *Robert Mayer*, for example, was ultimately proved right in his conviction, for which he fought fanatically. Similarly, *Gregor Mendel's* discovery—which is of far greater practical significance than, say, that of Copernicus—is in no way diminished by the fact that it was achieved by a man suffering from mental distress. On the other hand, *Goethe's* theory of colours, for instance—which he regarded as the chief achievement of his life and which he defended with pathological tenacity—is untenable.

In families of brilliant individuals, mental disorders are found almost as a matter of course among other members as well. *Goethe's* father, for example, was clearly psychopathic; *his* sister was severely so, as was his son. Research into the idiotypical determinants of genius must therefore also take thorough account of pathological predispositions within the family, as Möbius first taught.

> For most people, it is an uncomfortable thought that the achievements and deeds of those men whom they are accustomed to venerating as heroes of the mind might, in part, stem from a pathological predisposition. It should therefore be reiterated that illness means nothing more than a comparatively low degree of conformity to the principles of preservation. As long as the concept of illness is oriented towards the preservation of the individual, one cannot avoid acknowledging a connection between genius and illness. However, the concept of illness can also be related to the preservation of the race, and in that case the question takes on a different aspect.
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> Ultimately, all organic adaptation is directed not towards the preservation of the individual, but towards that of the race. There are organs and functions—such as those involved in reproduction—which serve not the preservation of the individual at all, but only that of the race. On the other hand, all organs and functions that enable the preservation of the individual also serve the preservation of the race. The preservation of the race is therefore the overarching concept in relation to the preservation of the individual. Just as with the concept of adaptation, the related concepts of illness and health must therefore also be oriented towards the preservation of the race. Thus, for example, infertility, whatever the reasons for its occurrence, is undoubtedly a pathological condition, even though it does not impair the preservation of the individual. On the other hand, childbirth and the postnatal period, although they inevitably entail certain risks for the woman, are not to be regarded as pathological but as entirely normal.
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> Similarly, the creative activity of a genius, even if it impairs individual survival, can nevertheless serve the life of the race. And such a disposition would be life-affirming in the highest sense, and thus fundamentally healthy. After all, not all people need to conform to the average. A population consisting entirely of geniuses would, of course, be hardly viable ; yet individuals can achieve the very highest for the life of the race, and it is quite literally a matter of life and death for a race that it continually produces men who open up new paths of life for it.

4. Race and Talent.

In the second section, it was shown that the various hereditary traits which are expressed in a person's physical appearance occur with very different frequencies in different regions; for example, light-coloured eyes, which are found in the vast majority of the population in Northern Europe, become increasingly rare the further south and east one goes, until they no longer occur at all in Central Africa or East Asia

. In a similar way, mental hereditary traits are also distributed differently across different countries. Traits that exhibit such varying distribution are precisely what are termed racial traits, and there is no fundamental difference between them and other hereditary traits, not even in comparison with pathological ones. The differences between the various racial predispositions are, however, generally less pronounced than those between normal and pathological predispositions; thus, the psychological difference between a Chinese person and a Black person is smaller than that between a normal Chinese person and a mentally deficient one. On the other hand, however, a European born with the psychological constitution of an Australian aboriginal would probably be unable to hold his own in the competitive demands of modern life and would presumably be regarded as mentally deficient, just as, for example, a Black person born with the lack of pigmentation typical of a Nordic person would have reduced resilience in his tropical homeland and would rightly be regarded as albino, i.e. pathological. The various races are, in general, simply adapted to their native environment, and these differences in adaptation bear a certain relation to the distinction between health and illness, which is itself likewise based on adaptation.

Although racial differences are generally far less pronounced than those between normal and markedly pathological dispositions, they are no less significant for that, precisely because they concern the differences between entire large groups and populations. This applies particularly to psychological differences. If there were only physical racial differences, the whole question of race would be of no particular significance; and it is evidently connected with this that it is precisely the psychological racial differences which are, as a rule, either exaggerated or denied altogether. There can be no doubt from the outset that psychological racial differences exist at all. After all, every race exhibits certain average characteristics in the structure of every organ; this naturally also applies to the structure of the brain and, consequently, to mental dispositions. The question, therefore, cannot be whether mental racial differences exist at all, but only what their nature is and how great they are.

The fact that mental dispositions are hereditary has already been discussed at length. However, these dispositions are, of course, not something abstract that floats, as it were, in the air, but are attached to organic forms, to the various idiotypes. After all, heredity consists precisely in the fact that organic forms retain their distinctive characteristics. Hereditary traits are racial traits; this also applies to psychological hereditary traits. A very large proportion of the psychological differences we have discussed are likely even to be based on racial differences in the narrower anthropological sense. After all, the individual racial traits are preserved even within a mixed population; and just as we attribute common hereditary characteristics of the body in different individuals to a common ancestry, so too do common traits of the soul stem from a common origin. The question therefore arises as to the psychological characteristics of those original races from which modern mixed populations have emerged.

Naturally, these can only be average values. Nor, of course, were the original races ever entirely uniform in themselves, but were always composed of a large number of idiotypes that differed slightly from one another. Furthermore, in every race there occasionally occur such significant genetic changes in individual idiotypes that these are now further removed from the original race than the original race is from other races; this applies, for example, to those idiovariations that cause mental deficiency or other pathological predispositions. However, such markedly deviant predispositions are, for the most part, soon eliminated again by natural selection, and insofar as they persist for a shorter or longer period, they cannot, nevertheless, negate the average differences between the races.

In a mixed population such as ours, one cannot simply infer a person's racial psychological predispositions from their physical racial characteristics. It is, for example, entirely possible that a fair-haired person with light-coloured eyes might have a psychological disposition more typical of a dark-skinned race. One can draw conclusions about a person's racial psychological predispositions from their ancestry with greater certainty than from such physical racial characteristics. Among Lower Saxon farmers, a small, short-headed, dark-haired person is an exception; yet they are still much

greater likelihood of possessing Nordic psychological traits than, for example, a tall, fair-haired, long-headed Italian. Since most people do not represent such exceptions, but resemble the average physical and psychological profile of their group, a conclusion drawn from physical appearance regarding psychological disposition is very often at least approximately accurate; and in practice, indeed, all people attach more or less importance to it.

Of course, we can no longer directly ascertain the psychological differences between the original races that have been incorporated into our mixed population. We must therefore content ourselves with inferring the psychological dispositions of the original races from the psychological differences observed in populations in which we simultaneously perceive a varied mixture of physical racial traits. It is, however, problematic to attempt to deduce the dispositions of the races from particularly 'typical' representatives, because the selection of the
'typical' individuals already presupposes certain preconceptions about the psychological character of the race. *Goethe*, in particular, is a popular 'type' on the basis of which such 'racial researchers' construct their image of the race. It is much easier to identify racial psychological dispositions in groups that are little or not at all mixed—such as East Asians, and certain African and Native American tribes—than it is within our own population. The cultural achievements of the various groups, in particular, allow valuable conclusions to be drawn.

That group of primitive races, to which the Wedda and the aborigines of Australia belong, is evidently still relatively closest to the ape-like ancestors of humankind, even in psychological terms. The members of these races earn their livelihood by roaming through the forests and consuming whatever edible food they find. The production of food through livestock farming or agriculture plays no part in their lives. Even when coming into contact with civilised peoples, these races have been able to adopt little or nothing of their culture; instead, they perish as a result.

Compared with these races, the Negroes live much less from hand to mouth. They have developed a fairly extensive system of livestock farming and, to a certain extent, crop cultivation. They have also developed certain trades, such as blacksmithing. Compared with European races, however, a lack of foresight is evident among

Negro. The prospect of future prosperity generally fails to motivate him to work persistently. The Negro is far more attuned to immediate sensory impressions than the European; he is therefore easily swayed by trinkets. Depending on his immediate experiences, he fluctuates between carefree exuberance and helpless despondency. During childhood, however, the Negro initially keeps fairly even pace with children of European races. Nevertheless, studies of American primary school pupils have consistently shown that, in psychological tests, Negro children's performance lagged behind that of 'white' children by about 30 per cent. With the early onset of sexual maturity, however, a stage is reached beyond which the Negro does not progress significantly. Black people have not produced any social structures comparable to those of Europeans or Asians; Black states such as Haiti or Liberia are characterised by mismanagement. Where Black people live alongside people of European descent, as in the southern states of North America, they are invariably found only in the lower strata of the population, engaged in menial occupations. No Black person has ever produced any works of genius.

On average, the Mongoloid races considerably surpass Black people in intellectual ability. Among the East Asian Mongols, agriculture and livestock farming have reached a high level of development over millennia. The formation of the Chinese Empire and its survival over five millennia bear unequivocal witness to the Mongols' great talent for building societies. The Japanese and Siamese empires, too, were founded by Mongols. In the Americas, the ancient Peruvian and ancient Mexican empires were established by related races.

That said, among the Mongoloid races, the greatest cultural aptitude clearly belongs to the East Asians. Among the Mongols, the dispositions geared towards social life are particularly well developed. On the whole, they possess a greater capacity for imitation than for invention. Their memory is stronger than their critical faculties. They therefore have little interest in the abstract sciences, but tend to revere history. Whilst the Indian Aryans, who achieved such excellence in philosophy and architecture, have completely

, the Chinese possess meticulous historical and genealogical records spanning millennia. Genius thinkers, inventors and discoverers in the European sense are scarcely to be found amongst the Mongols. The remarkable consistency which, despite occasional vicissitudes, characterises the history of Chinese culture is certainly, to a large extent, a consequence of the Mongolian character, which tends to cling to tradition. The Mongol's metaphysical needs are as limited as his scientific ones. The teachings of Confucius and Laozi do not deal with metaphysical matters, but are entirely focused on practical social life. Several religions coexist in East Asia in a spirit of mutual tolerance. The history of East Asia knows no religious wars such as those that have torn Europe apart. It is consistent with this practical sobriety and limited development of the imagination that the Mongol is also unfamiliar with romantic love in the European sense. Contrary to the notions we tend to associate with the terms 'Huns' or
'Tatars', it is evident that the Mongoloid races as a whole are certainly less savage and warlike than the truly European ones. They differ from the latter, both mentally and physically, in much the same way as a woman differs from a man; they are more receptive than creative, whilst being frugal and patient. Their great frugality and resilience enable them to thrive even in meagre living conditions to which the truly European races are unable to adapt.

It is difficult to say anything definite about the Mongoloid races of Europe, because they occur only in mixture with others. Nevertheless, it can be said that European populations with a strong Mongoloid influence differ from those of predominantly Nordic stock in much the same way as the Mongol differs from the European. Mongoloid racial elements have certainly played a significant part in the various cultures that Europe has experienced over the millennia, although they cannot, of course, be described as the actual creators of any of these cultures.

Peoples of predominantly Armenianoid race (cf. p. 134), such as Armenians, Greeks and Jews, are characterised by a particular aptitude for business in trade and commerce, for which they are endowed, in addition to a

high degree of intelligence, above all the ability to empathise with the inner lives of other people. The Armenoid race has, in any case, made an outstanding contribution to Assyrian-Babylonian and ancient Jewish culture.

The Mediterranean race is characterised, in contrast to the calm, staid nature of the Mongoloid race, by a certain restless wildness. The psychological differences between the populations of Sicily, Corsica and North-West Africa, for example, and those of Eastern Europe are striking. The Mediterranean people lack the calm industriousness of the Mongoloid; they also seem to be somewhat inferior to them in intellectual ability.

The Oriental race, on the other hand, which is related to the Mediterranean race, is characterised not only by intelligence but also by energy and a spirit of enterprise. It has even less inclination towards the calm, steady work of the farmer; rather, it has a marked tendency towards nomadism. It has also produced daring seafarers such as the Phoenicians. Arab tribes form a ruling class in a large part of Africa. The Oriental race may be regarded as the driving force behind ancient Egyptian civilisation, as well as Phoenician and Punic civilisation, ancient Jewish civilisation and the Arab civilisation of the Middle Ages. It also played a significant role in Assyrian-Babylonian culture. Its influence on modern Western culture is also very profound, precisely because it forms an essential part of the Jewish population.

However, it was the N o r t h e r n r a c e that brought the actual movement into European history. The Indo-European cultures originated from it. Thus, the culture of ancient India is an unmistakable consequence of the conquest of the land by the Northern Aryans, who invaded it in the second millennium BC. The power and splendour of the ancient Persian Empire rested on the shoulders of another branch of the Aryans. The incomparable culture of ancient Greece followed the immigration of the Aryan Hellenes, whose Nordic race is attested not less by their surviving works of art than by their historical records. The Roman Empire had its roots in the Celtic Italics, who, like the other ancient Celts, are likely to have been predominantly of Nordic race. The Germanic kingdoms, which emerged from the so-called Migration Period

, were founded by tribes of the Nordic race. The German Empire of the Middle Ages rested entirely on the shoulders of the Germanic peoples. Vast coastal regions of the Mediterranean were ruled by seafaring Normans. The blood of the Lombards is evident in the Italian Renaissance. No Renaissance flourished on the territory of the Eastern Roman Empire, which was conquered not by Nordic tribes but by the Mongoloid Turks. The Iberian Peninsula, which, in comparison with the other two southern peninsulas, had experienced only minimal Nordic immigration in early historical times, did not experience such a cultural flowering in antiquity either. However, after the Visigoths had arrived in Spain, a Spanish empire arose in the late Middle Ages in which 'the sun never set'. The Nordic influence also evidently played a decisive role in the Portuguese discoveries and conquests. The Reformation was born of the distinctive nature of the Nordic spirit, and on the whole it took hold only in countries with a predominantly Nordic population. The small Dutch nation achieved outstanding feats at sea and established a vast and flourishing colonial empire. The small Swedish kingdom, under Gustavus Adolphus and Charles
XII, it displayed tremendous political vigour. The French power of past centuries rested upon the descendants of the Franks, Goths and Normans. The great Russian Empire was founded by Normans (Varangians). The Anglo-Saxon colonisation of North America, South Africa and Australia over the last three centuries, driven by the descendants of Saxons and Normans, represents the most massive wave of expansion of Nordic blood since the Migration Period, and perhaps the most massive in world history altogether. Spanish and Portuguese South America, whose ruling class—unlike that of North America—is not predominantly Nordic but Mediterranean, cannot compare with the latter in terms of economic and cultural significance.

Modern Western culture is by no means evenly distributed across the globe; rather, it is mainly at home in countries with a predominantly Nordic population or those which at least have a strong Nordic influence. The great scientific discoveries, inventions and other intellectual achievements of the present day almost all originate either from the north-western half

of Europe (including Finland, Austria and Switzerland) or from North America.

If we consider the intellectual leaders of mankind—the great statesmen and military commanders, researchers and philosophers, inventors and explorers, artists and poets—in terms of their physical type, we find that the vast majority are, in their appearance too, predominantly of the Nordic race. This observation has become so ingrained in the general consciousness that, for example, even Christ is regularly depicted as a man of the Nordic type. Even during the World War, the most successful leaders on both sides were of the Nordic race: not only *Hindenburg* and *Ludendorff*, but also *Joffre* and *Foch*, *Lloyd George* and *Wilson*.

Of all people, the Nordic individual is the least given to living in the moment; he surpasses all other races in steadfastness of will and prudent foresight. As a result of this forward-thinking disposition, sensual impulses are subordinated to more far-reaching goals. He is also undoubtedly superior to the Mongoloid, if not in terms of receptive intelligence, then certainly in terms of the creative power of the mind. However, this too applies only to the average; for there are certainly many gifted Mongoloids who can certainly hold their own against most people of the Nordic race.

The psychological character of the Nordic race is evidently linked to the Nordic environment, but not in the sense that the damp, cold climate directly produced their provident disposition; rather, it is in the sense that families with the carefree disposition of southerners, who do not habitually plan far ahead, perished much more frequently in the Nordic winter. The race is therefore, in a certain sense, the product of its environment, but not the direct product of the environment in the Lamarckian sense; rather, it is the product of selective adaptation to the environment. Of course, the original developmental potential of a race is also of essential importance in this regard. Mongoloid races, too, have been adapted to the northern climate through natural selection. However, whilst the Nordic race overcame the inhospitable conditions of its environment by enhancing its intellectual faculties, the adaptation of the Arctic Mongoloids was achieved through the development of extreme frugality.

The Nordic man is often contrasted with the roving nomad as a settled farmer. However, he is less of a typical farmer than the Mongoloid. One might rather regard the Viking as the archetype of the Nordic man. Daring and strong-willed to the point of ruthlessness, he is capable of devoting himself to a cause without regard for others. This is linked to the fact that he finds it difficult to empathise with other people's feelings. Stubbornly, he insists on having his own way. Whilst his talent for organisation makes him of great importance to social life, he fundamentally despises community. His instincts are more individualistic than socially oriented. Whilst the Eastern Mongols have been living together in an empire numbering several hundred million for almost 5,000 years, the Nordic peoples have divided themselves into numerous small groups

and have repeatedly torn one another apart. Their exaggerated self-confidence stands in the way of a unifying alliance today, just as it has for millennia.

The relatively limited musical talent of the Nordic people may be linked to this lack of empathetic understanding. The Indian Aryans, the Hellenes, the modern Scandinavians and the Anglo-Saxons have achieved great things in the visual arts, but not in music. 'Frisia non cantat.' Italians, Magyars, Jews and Gypsies are regarded by the Germanic peoples—and probably not without reason—as being particularly musical.

The disposition towards anxious foresight compels the Nordic people to ask about the origin and destiny of the world and of humankind. They are, in essence, the truly religious and philosophical people. The Nordic longing finds no satisfaction; it therefore easily soars beyond all reality and turns to the metaphysical. The Nordic people also tend towards a peculiar spiritualisation of love, which can easily take on an unhealthy character and become a danger to the life of the race.

It is very common to contrast the racial disposition of the Germanic peoples with that of the Jews, although it is usually overlooked that neither group constitutes a homogeneous race. On the other hand, of course, the psychological character of the Jews cannot simply be explained by the races from which the Jewish community emerged. The conditions of natural selection during many centuries of urban life and the almost exclusive engagement in trade and similar occupations were evidently of the most significant influence. Excluded from the primary production of the necessities of life, not only by their own inclinations but in many cases also by legal restrictions, it was generally only those Jews who were particularly adept at winning over members of the host populations who were able to start a family. This may well explain why Jews distinguish themselves not only through cleverness and enterprise, diligence and perseverance, but above all through their ability to put themselves in other people's shoes and guide them according to their own will. Their inclinations and abilities therefore repeatedly lead them to pursuits in which responding to the respective inclinations of the public and guiding them brings success. The professions to which they turn with a preference and with success are therefore, above all, those of merchant, trader and moneylender, journalist, writer, politician, actor, musician, lawyer and doctor. Famous Jewish scientists can be found in the fields of medicine, mathematics, physics and psychology. Almost all the great chess masters are Jewish. Their appreciation of nature is strikingly limited. Jews are also generally not particularly gifted in the visual arts. Jews usually have a strong aversion to physical labour. They generally fear pain, illness and death more than Germans do. Nor do they generally have a particular fondness for military service.

On the other hand, one must not overlook the fact that the Germanic peoples and the Jews are quite similar in their fundamental dispositions; this is particularly true if one defines 'Germanic peoples' as comprising only those of predominantly Nordic race. Both are characterised by high intellectual ability and strength of will; both possess great self-confidence, an enterprising spirit and a pronounced will to dominate. In terms of business acumen, the Jews are somewhat outdone by the Nordic Hanseatics, Scots and Yankees. The hostility between the Germanic peoples and the Jews, which manifests itself in many instances, is likely to stem at least as much from the similarity in their abilities—which leads to fierce competition—as from their differences in character, much as the various Germanic ruling groups repeatedly find themselves embroiled in power struggles.

Unfortunately, the racial-psychological analysis is time and again conflated with moral judgement. The Jewish disposition is, in and of itself, by no means directed towards negation and subversion. Even where he destroys, he generally intends to build. The

The fact that the leaders of radical revolutionary movements are mostly Jews can be explained rather by dispositions of the Jewish soul which, from a biological point of view, are neither good nor evil. The ability to empathise with the moods of others, to put oneself in their shoes, makes the Jew not only a natural actor, but also a natural orator and demagogue. Jewish organisers (see p. 234) play the greatest role in modern revolutionary movements because they are able to immerse themselves completely even in utopian ideas and can therefore make convincing promises to the masses with a high degree of inner sincerity.

The pronounced sense of family among Jews is anything but corrosive, as are their strong sense of solidarity, their mutual willingness to help one another, and their sense of universal humanity. The Jewish spirit, alongside the Germanic spirit, is the principal driving force behind modern Western culture. The emancipation of the Jews has had a similar effect to that of one of the waves of Nordic blood on the Indo-European cultures. But the Jewish spirit has also been unmistakably influential in the development and spread of Christianity as one of the most essential roots of Western culture.

It goes without saying that the inherited racial traits are not the sole cause of the great cultural achievements. All culture, all achievement arise far more from the interaction of racial predisposition and environment. The racial-biological perspective must take care not to fall into similar one-sidedness as certain widespread doctrines that oppose it. Thus, the so-called 'materialist' view of culture, which draws on the name of Marx, teaches that economic conditions alone determine all culture; it also seeks to trace all moral and intellectual ideals back to economic conditions. The so-called 'idealistic' view, by contrast, regards precisely intellectual achievements and ideals as the actual driving forces of history; yet in its common form, as espoused, for example, by Natorp, it denies—just as the materialistic view does—that race plays a significant role. Common to both is the Lamarckian fallacy of an almost unlimited malleability of human beings. Whilst the 'materialist' view overestimates malleability through the direct influence of the environment, the 'idealist' view dreams of the ennoblement of the human race through the internalisation of intellectual ideals; yet this, too, presupposes the heritability of acquired adaptations and is therefore untenable. Human intellectual disposition cannot simply be reshaped by either economic or intellectual influences, but only through selection; it is a racial predisposition, and all intellectual

achievements depend first and foremost on the hereditary predisposition of their creators and bearers. An organic conception of culture must not, admittedly, overlook the conditions emphasised one-sidedly by the 'materialist' and the 'idealist'; yet, in contrast to these, it must point to race or, more generally, to hereditary predisposition as the primary and most indispensable condition of all culture. Incidentally, the great Kant, to whom modern 'idealists' are so fond of referring, refrained from such one-sidedness and emphasised that the

'innate character lies in the mixture of human blood, and even the acquired and artificial character is merely a consequence of this'.

It is a widely held view that, in cases of racial mixing, the mental disposition of the inferior race invariably prevails; indeed, that mixed-race individuals are even inferior to both parent races. Thus, it is reported of the mixed-race populations in the coastal regions of Africa, Central and South America that they are morally inferior to pure-blooded Blacks or Indians, whilst in terms of intelligence they are their equals or superior. It is likely that the inferiority of these mixed-race individuals is largely the result of natural selection. The white men who father mixed-race offspring with native women represent an unfavourable selection in terms of character, and the native women who consent to relations with white men are, of course, often of loose character; it is no wonder, then, that the mixed-race offspring are largely of the same sort. Where the conditions for natural selection are more favourable, there is no inferiority of the mixed-race individuals compared with the indigenous race. This applies, for example, to the mixed-race people of Rehoboth described by Eugen Fischer, who are the descendants of Dutch colonists and Hottentot women whom the former took as lawful wives. Fischer found that the bastards are superior to the pure-blooded Hottentots not only in intelligence but also in character, whilst, on the other hand, they are considerably inferior to the Boers in terms of cultural aptitude.

In any case, it is not true that great cultural achievements can be accomplished only by pure races, nor, of course, is it true that the purity of a race is any more important than the nature of its innate aptitude. The Nordic tribes that migrated to ancient India, Greece and Italy only reached their greatest splendour at a time when extensive intermingling with the indigenous population had already taken place. Some of the greatest minds, such as *Michelangelo*, *Luther* and *Beethoven*, were evidently of mixed descent. *Goethe*, too, was evidently neither physically nor mentally of a purely Nordic type; judging by the portraits, this different influence is likely to have stemmed primarily from his maternal grandmother, who was a *Lindheimer* by birth.

Of course, in many cases racial mixing can also lead to unfavourable combinations. The individual traits of each race have been adapted to one another through natural selection over the course of countless generations, and mixing can disrupt this harmony. But even a mismatch between different traits need not always be unfavourable in terms of cultural creation. As discussed above, it is precisely disharmonious natures that are occasionally capable of great cultural achievements. Gobineau, to whom advocates of racial purity often refer, explicitly spoke of 'the world of the arts and of higher literature as the result of the mixing of blood' and said: 'It would be wrong to claim that all mixtures are evil and harmful.' On the other hand, he certainly knew of no equal mixture for the Nordic race, and he saw

the downfall of the race and culture lay in the fact that the noble race, being fewer in number, would gradually be swallowed up by the flood of the ignoble through intermingling.

The cultural aptitude of the various races is discussed so extensively and contested so passionately precisely because it is believed that the value of the races depends upon it. We, too, have hitherto, when we have described certain racial characteristics as valuable and others as inferior, taken cultural aptitude as our yardstick, deliberately drawing on commonly held views. From a philosophical standpoint, however, this yardstick is neither self-evident nor unambiguous. For if one wishes to measure the value of races by their culture, the question of the meaning and value of culture itself inevitably arises. Is, for example, Western culture superior to Chinese culture? As will be shown in the second volume, Western culture is currently taking a direction which amounts to the eradication of its creators and bearers, and thus to its own destruction. One might be inclined to say that only a healthy culture should be taken as the standard for racial value. Would that mean, then, that Chinese culture—which is undoubtedly more conducive to the survival of its bearers—is therefore the superior culture, and its creators the superior race? We shall not be inclined to draw this conclusion, and indeed it would amount to nothing more than a circular argument. For if one measures the value of a culture by its effect on the race, one cannot in turn measure the value of the race by its effect on the culture. But if one recognises the race as having intrinsic value in its own right, then a race can be neither 'higher' nor
'lower' than another, because all such comparisons of superiority would presuppose the validity of a separate standard. The Earth is neither higher nor lower than, say, Mars, nor is it at the same height, because the concepts of high and low are oriented towards the Earth itself. The same applies to race. If we love our race not for the sake of any particular culture, doctrine or moral code, but for its own sake, then this love is incompatible with holding any other race in equal esteem, without our consequently regarding our own race as superior in an objective sense.

Bibliography for the first volume.

As this book is not intended to be an exhaustive handbook, we have refrained from providing a complete bibliography, which would be exceptionally extensive and would make the book unnecessarily expensive. Instead, we have confined ourselves to citing the most important works. To assist non-specialists, we felt it important to include brief critical comments alongside the titles of the books. In the case of books containing detailed bibliographies, this has been indicated in order to guide readers who are interested in the specific literature of a particular sub-field.

On the first section.

The author of the first section has presented the theory of heredity in greater detail in a separate book:

Baur, E. An Introduction to Experimental Genetics. 3rd and 4th editions. Berlin 1919. (Contains a 30-page bibliography of the most important literature in the field of genetics.)

Other German textbooks on genetics include:

Johannsen, W. *Elements of Exact Genetics*. 2nd ed. Jena 1913. (Highly recommended, particularly as an introduction to biological variation statistics. However, we cannot fully agree with the discussion on the significance of natural selection.)

Goldschmidt, R. Introduction to the science of heredity. 3rd ed. Leipzig 1920. (Very comprehensive; however, our views differ from those of the author on certain points. Includes bibliographies at the end of each chapter.)

Plate, L. The Theory of Inheritance. Leipzig 1913. (Deals with the inheritance of human traits, including pathological ones, in greater detail than the other textbooks. However, the views presented differ

those we consider to be correct, in some cases considerably. Includes a bibliography.)

Haecker, V. General Theory of Inheritance. 2nd ed. Braunschweig 1912. (Considers the relationship between the theory of inheritance and cell research in particular detail. Includes bibliographies at the end of each chapter. The 3rd ed. of 1921 was not yet available to us.)

Ziegler, H. E. The Theory of Inheritance in Biology and Sociology. Jena 1918. (Also deals with biological and social selection. We cannot fully agree with Ziegler's views on heredity on only a few points, e.g. regarding the function of chromosomes.)

Bateson, W. Mendel's Theories of Inheritance. Translated from the English. Leipzig and Berlin 1914. (Also contains a description of Mendel's life and a reproduction of his classic work from 1865.)

Siemens, H. W. The Biological Foundations of Racial Hygiene and Population Policy. Munich 1917. (This inexpensive little booklet is particularly recommended as an introduction to the study of heredity and racial biology.)

The leading journals in the field of heredity research are:

Journal for Inductive Descent and Genetics. Edited by E. Baur. Published by Bornträger, Berlin.

Archive for Racial and Social Biology. Editor: A. Ploetz; published by Teubner, Leipzig.

The former deals more with conditions in plants and animals, the latter with those in humans. Both publish regular literature reviews and critical discussions.

Journal of Genetics. Edited by W. Bateson and R. C. Punnett. Cambridge. (The most important English journal for research into heredity.)

Journal of Heredity. Editor: P. Popenoe. Washington. (Leading journal in America.)

Hereditas. Editor: R. Larsson. Lund. (This Scandinavian journal of heredity research also publishes many papers in German or English.)

On to the second section.

Martin, R. Textbook of Anthropology. Jena 1914. (Martin's work, which is more of a handbook than a textbook, presents anthropological methodology and the variation of individual human characteristics and organs with a comprehensiveness otherwise unrivalled. The 100-page bibliography may be regarded as exhaustive.)

Ripley, W. *The Races of Europe*. 2nd ed. London 1912. (This fine work by the American researcher provides the best description to date of the racial composition of Europe, illustrated with numerous images.)

Deniker, J. *The Races of Man*. London 1900.

— — *Les races de la terre*. Paris 1914. (Deniker's work is fundamental in many respects. However, his division of the major races seems to us to go too far. He may, in some cases, have referred to racial mixtures as races.)

Fischer, E. The articles 'Anthropology', 'Anthropogenesis', 'Fossil Hominids', 'Brain', 'Hair, Skin', 'Body shapes', 'Races', 'craniology', "Social Anthropology" in the Concise Dictionary of the Natural Sciences. Jena 1912/13. (In these articles, the author of the 2. section of this book has presented his anthropological views in concise form. Specialist literature is also listed there.)

Fischer, E. The Rehoboth Bastards and the Problem of Hybridisation in Humans. Jena 1913. (The relevant specialist literature is also listed there.)

Retzius, G. and **Fürst.** *Anthropologia suecica*. Stockholm 1900. (A classic monograph on the racial composition of Sweden.)

Ammon, O. On the Anthropology of the People of Baden. Jena 1899. (The sole and fundamental monograph on the racial composition of a German state.)

Giuffrida-Ruggeri, V. *Homo sapiens*. Vienna 1913. *L'uomo attuale*. Milan 1913.

Sergi, G. *Europa*. Turin 1908. (Sergi's views differ, in some cases quite significantly, from those we consider to be correct.)

Klaatsch, H. *The Development of Humanity*. Berlin 1921. * * (We, too, can only partially agree with Klaatsch's views.)

Guenther, K. *From Primitive Animal to Human*. Stuttgart 1909. (With a bibliography.)

Haecker, V. An Analysis of Evolutionary Characteristics. Jena 1918. (Relevant literature also cited therein.)

On the third section.

The author of the third section had already set out his fundamental biological views and their implications for the field of pathology in a separate treatise in 1912:

Lenz, F. On the Pathological Hereditary Factors in Men and the Determination of Sex in Humans. Jena 1912. (Some details of this work, which sought to cover too much ground, are now outdated. The author is, however, pleased to note that all the fundamental views expressed there have since largely gained acceptance

Since 1919, he has also set out his views in his lectures on racial hygiene. The present outline constitutes, in part, an extract from these lectures.)

— — On Dominant Sex-Linked Inbreeding. Archive for Racial and Social Biology 1918/19, Vol. 1. (In this paper, the author analysed for the first time the difference between dominant and recessive sex-linked genetic traits.)

— — The Significance of the Statistically Determined Degree of Consanguinity among Parents. Munich Med. Wochenschrift 1919 No. 47. (First demonstration that, in the case of recessive disorders, the rarer the disorder in question, the higher the percentage of consanguinity among the parents that can be expected.)

Siemens, H. W. Introduction to General Constitutional and Combination Pathology. Berlin 1921. (The fundamental concepts and also the vast majority of views on specific details in this book are entirely consistent with our own. As we only became aware of the book's publication after the proofreading of our text had been completed, it could not have exerted any influence on it. The agreement is explained not only by the general evidence of the facts and the impact of earlier writings, but also by an intensive personal exchange of ideas. The book also contains a selected bibliography of 67 entries.)

Bauer, J. The Constitutional Disposition to Internal Diseases. 2nd ed. Berlin 1921. (An exceptionally diligent compilation, which, admittedly, essentially still follows the lines of older constitutional pathology prior to the advent of modern research into heredity. The extensive bibliography, comprising 88 pages, is quite valuable; however, it has been compiled somewhat haphazardly and therefore does not allow for any distinction between writings of greater or lesser value.)

Brugsch, Th. General Prognostics. Berlin 1918. (This book, too, contains much of value.)

Martius, F. Constitution and Heredity in their Relations to Pathology. Berlin 1914. (The author of this work has earned lasting credit by emphatically highlighting the importance of hereditary predisposition at a time when external causes of disease were being one-sidedly overestimated, albeit in a pre-Mendelian spirit. We cannot agree with him on the details.)

Dresel, K. To what extent do Mendel's laws of inheritance apply in human pathology? Virchow's Archive of Pathological Anatomy. Vol. 224. 1918. (Contains a valuable bibliography.)

Diseases and Marriage. 2nd ed. Revised and edited by C. v. Noorden and S. Kaminer. Leipzig 1916. (This anthology contains a variety of valuable material on the significance of pathological hereditary factors, particularly in the chapters on diseases of the bones and joints [C. Helbing], on eye disorders [G. Abelsdorff], on

metabolic disorders [C. v. Noorden] and on speech disorders [H. Gutmann]; in other chapters, the question of heredity is dealt with less thoroughly; the bibliographies for the individual chapters are correspondingly of varying quality.)

Treasury of Human Inheritance. (Published by the Institute of Racial Hygiene [Galton Institute] at the University of London, headed by K. Pearson. The Treasury is published in instalments as individual volumes, each of which deals with a hereditary disease or a group of such diseases. The individual volumes constitute unrivalled, comprehensive collections of the family trees known to date and the relevant literature.)

Bulletins of the Eugenics Record Office. (These issues from the Institute of Racial Hygiene at Cold Spring Harbor in the State of New York, directed by C.
B. Davenport at Cold Spring Harbor in the State of New York, the heritability of pathological predispositions is also frequently addressed in valuable individual studies.)

Comprehensive American accounts include:

Davenport, C. B. *Heredity in Relation to Eugenics*. New York 1911.

Popenoe, P. and **Johnson, R. H.** *Applied Eugenics*. New York 1920. (This excellent work, accompanied by careful bibliographical references, is warmly recommended as an introduction to the American literature on our subject.)

As sources of information on the heritability of pathological predispositions, reference should once again be made to the journals already mentioned in the first section, in particular the ***Archiv für Rassen- und Gesellschaftsbiologie***. However, most reports on the hereditary nature of certain conditions appear in the specialist journals of the individual medical disciplines, which cannot be listed here individually. The journal review and the research papers section of the *Archiv für Rassenbiologie*, as well as the journal review of general medical journals, such as e.g. the *Münchner Medizinische Wochenschrift*.

For guidance on the existing literature concerning hereditary disorders of the individual organ systems, particular attention should be drawn to the following works:

Groenouw, A. Relationships between general ailments and organ diseases to changes and diseases of the visual organ. As a volume of Graefe and Saemisch's Handbook of Comprehensive Ophthalmology.
3. ed. Berlin 1920. (Gronoouw's book contains a thorough and detailed account of hereditary eye disorders and the relevant literature.)

Steiger, A. The Origin of Spherical Refractive Errors of the Human Eye. Berlin 1913. (Particular mention should be made of this groundbreaking work by Steiger, which has had a truly liberating effect on the question of the aetiology of myopia.)

Stein, C. The Auditory Organ and Constitution. Journal of Otology. Vol. 76, 1917. (Contains a valuable bibliography on hereditary ear disorders.)

Bettmann, S. Skin Malformations. Jena 1912. (A detailed bibliography on hereditary skin disorders.)

Meirowsky, E. On the Origin of the So-called Congenital Malformations of the Skin. Vienna and Leipzig 1919. (Contains a valuable collection of family trees and references.)

For guidance on the literature in the field of malformations, constitutional anomalies and internal disorders, reference should be made to the more general works already mentioned. The following should also be noted:

Toenniessen, E. Hereditary Research and Internal Medicine. In: Results of Internal Medicine and Paediatrics. Vol.
17. 1919. (Contains no specific references.)

Pfaundler, M. v. On the Nature and Treatment of Diatheses in Childhood. Proceedings of the 28th Congress of Internal Medicine. Wiesbaden 1911.

Pfaundler, M. v. Childhood Predispositions to Disease (Diatheses) and Probability Calculations. In: Journal of Paediatrics. Vol. 4. 1912.

Jendrassik, E. Hereditary Diseases. In Lewandowski's Handbook of Neurology. Vol. 2. Berlin 1911. (We can only partially agree with the author's views on hereditary nervous disorders. Contains a valuable bibliography.)

Kraepelin, E. *Psychiatry*. 4 vols. Leipzig 1909–1915. (A comprehensive account of the entire field of mental disorders by the grand master of modern psychiatry. No bibliography.)

Rüdin, E. Studies on the Correlation and Origin of Mental Disorders. I. On the Association and New Development of Dementia praecox. Berlin 1916. (The author has made a groundbreaking contribution to the understanding of mental disorders within the framework of modern biology by undertaking research into the causes of

mental disorders, the author has made a groundbreaking contribution in the spirit of modern biology. With a bibliography.)

Lundborg, H. Medicinal-biological family studies within a farming family of 2,232 members in Sweden. Jena 1913. (This work, which reflects the immense effort of the renowned Swedish racial biologist, is the finest example of racial biology individual study of a distinct population group to have been published to date. The main focus is on hereditary nervous and mental disorders.)

The works by Rüdin and Lundborg also deal with the methodology of hereditary research. Furthermore, for guidance on the literature relating to Chapters 3 and 4 of the third section (pp. 239–266), reference must be made to the works cited for the first section.

On the fourth section.

Peters, W. On the Inheritance of Psychological Abilities. Leipzig 1915 (A fundamental work with carefully selected bibliographical references.)

Ziegler, H. E. The Theory of Correlation in Biology and Sociology. Jena 1918. (We would like to draw particular attention once again to this work, already mentioned at the outset, as our discussion of the heritability of intellectual ability is partly based on it.)

Schallmayer, W. Heredity and Selection. 4th ed. Jena 1920. (Although this comprehensive work is also relevant to the subjects covered in the earlier sections of our textbook, it is of particular importance to the fourth section. It also contains a meticulous and detailed bibliography. However, whilst the author denies the connection between anthropological race and aptitude, we cannot agree with him on this point.)

Galton, F. *Hereditary Genius*. London 1869. (The classic work on the heritability of talent. The German translation, published under the title *Genie und Vererbung* (*Genius and Heredity*), is unfortunately not entirely satisfactory.)

— — *Natural Inheritance*. London 1889. (Also a classic, although in some respects now out of date.)

Popenoe, P. and Johnson, R. H. *Applied Eugenics*. New York 1920. (This work, already mentioned, deals with the heritability of mental dispositions in a particularly skilful manner.)

Lombroso, C. The Genius. Hamburg 1900. (Although one-sided and outdated, Lombroso's book, first published in 1864, deserves mention here on account of its original significance.)

Moebius, P. J. *The Pathological Aspects in Goethe*. Leipzig 1898.

— — Nietzsche. Leipzig 1904. (Through these 'pathographies', Moebius has made a significant contribution to clarifying the relationship between psychopathy and talent. Unfortunately, there is as yet no book that truly deals comprehensively with this question in the spirit of modern biology and modern psychiatry.)

Fischer, E. Social Anthropology. In: Concise Dictionary of the Natural Sciences. Jena 1912/13. (This concise work also briefly outlines the cultural aptitude of the various races. I (Lenz) also owe valuable insights to the anthropological lectures of my teacher Eugen Fischer, without, however, wishing to attribute to him all the views developed in the fourth section.)

Ploetz, A. Social Anthropology. Special reprint from *Kultur der Gegenwart*. Leipzig 1920. Not yet available in bookshops. (I owe an immense debt of gratitude to Alfred Ploetz, the founder of German racial hygiene, whom I may well also describe as my teacher, for his insights into racial biology. His views on racial psychology, amongst other things, are also briefly expressed in the aforementioned 'Social Anthropology'.)

Gobineau, J. A. *An Essay on the Inequality of the Human Races*. Translated into German by L. Schemann. Stuttgart 1898. (Despite numerous errors in the details, this visionary work, which appeared in Paris in 1853–55, remains unshaken in its fundamental ideas; it has had an impact on the question of race like no other.

Woltmann, L. Political Anthropology. Eisenach 1903. (A significant continuation of Gobineau's teachings; though, biologically speaking, still rooted in the views of the pre-Mendelian era.)

Schemann, L. Gobineau's Work on Race. Stuttgart 1910. (A historical-critical defence of Gobineau's work, which on the whole may be described as successful.)

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BOOKS